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THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

ON MEDICAL ADVICE REGARDING THE MARRIAGE OF SUBJECTS WITH A PERSONAL OR FAMILY HISTORY OF INSANITY.¹

By HENRY R. STEDMAN, M.D.,

BOSTON.

IN considering this subject many questions arise which concern the general practitioner quite as closely as the alienist, as the family physician is almost invariably the first to be consulted in such matters, and his is often the only medical opinion asked.

It should be stated at the outset, that this paper is in no degree an effort to cope with the vast subject of the relations between insanity and marriage in general, but merely an attempt to state systematically what seem to me to be the salient considerations regarding medical advice as to the liability after marriage to insanity in the person or progeny of those who are hereditarily so predisposed or have experienced the disease in person. It concerns the solution of some of the most difficult problems in the practice of medicine.

That medical opinion on this subject is given comparatively little thought by people at large and by those who seek our advice in individual cases is evident from the great frequency of marriages in families of strong neuropathic

¹ Read before the Boston Society for Medical Improvement, Oct. 22d, 1888, and (by title) at the meeting of the Am. Neurological Assoc., Sept., 1888.

taint and the not uncommon union in wedlock of those who have been actually insane or possibly are so at the time. In the former class the physician's advice is rarely sought, and in the latter far too seldom. In both it is rarely heeded when given. When, in a given case, we believe our opinion to be sincerely sought and conscientiously followed, it is often more than likely that family, social or pecuniary influences have brought about the fortunate result. If, then, our counsel on this point is seldom asked, and when accepted rarely followed from disinterested motives, we can well understand why the subject receives such scant notice from physicians, and, with such a forbidding prospect, why it is looked upon as questionable whether the medical profession can be of much practical help in regulating such unions.

We can also, in a measure, explain the hastily considered advice often given in such cases, by the confidence that is felt that no matter what may be the physician's advice it will have little to do with the result.

We are by no means alone in the belief that the instances are comparatively few in which the man or woman who consults us is actuated by high motives and honestly regardless of personal disappointment will implicitly follow our advice in view of further and irreparable disaster to either contracting parties or their possible descendants. But when this is the case no physician can fail to be impressed by the enormous responsibility thrown upon him. And, by the way, can we invariably say that this or that case does not come under this head? If we cannot, there is another reason to be added to those now to be presented for the exercise of great caution in such consultations.

The causes just mentioned; the necessary lack of special knowledge of insanity in the profession at large; taken with the tendency to under-estimate dangers of the far future have been the prime factors in the present disposition to generalize rather than to definitely investigate individual cases as to their causes, nature and hereditary history. It is not a far-fetched criticism then that at least a little of the public indifference to medical opinion in such

matters may properly be laid at our door, so widely divergent and so inconsistent are the views of physicians in many cases which, by their very nature, could possibly result in but one, and that a disastrous way. Again, although the advice of the individual physician may have been a very small factor in promoting a marriage or in breaking an engagement, he is nevertheless frequently exposed to lasting blame or ridicule by the family according as the result proves to be disastrous or otherwise.

If these observations are accurate, we must conclude that careful examination of each case in all its bearings, based on the best knowledge to be obtained from the contracting parties, our own experience and that of others in the prognosis of mental disease, will lead to much practical help in these directions to physicians. If thereby special research in this comparatively neglected field of medical inquiry is developed, we may reasonably hope for some slight mitigation at least of this scourge to the individual, the family and society, through improved public sentiment born of a greater respect for medical opinion in such matters. Landmarks are therefore needed, especially for the guidance of the general practitioner, for to him alone in his double role of family physician and confidant such questions are, as we have said, often committed for decision.

A preliminary step in this direction is a knowledge of the obstacles which arise in the way of dealing with these problems. First come those of a social character, which sometimes present greater difficulties than the second class which relates to the purely medical aspect of the question.

It sometimes transpires in the course of our inquiries that on one side a family anxiously desires a marriage to take place for the sake of improving their relative's position socially or financially, or both, while in another case a union is opposed for the opposite reasons. Therefore, either important facts regarding the mental status of the party in question or his or her family history may be kept carefully out of sight, or trivial incidents may be greatly exaggerated, as the case may be. Thus the physician who feels called upon to give an opinion may, in a highly important case,

be obliged to obtain his own evidence unaided against a prearranged and formidable array of statements, and to pass judgment himself. It is well, then, in view of possibly calamitous consequences to be on our guard against such attempts to improperly influence our opinion. It is, therefore, permissible we think, without meddlesome or extended investigation to endeavor to ascertain the probabilities of interested evidence on both sides in order to clear the way for forming our judgment on purely medical grounds. If in cases involving commitment to an asylum it be necessary preparatory to the examination of the patient himself for the physician, before certifying, to learn from the family the truth regarding facts bearing upon the question of a person's insanity and to properly balance them to prevent his unjust commitment or any danger to himself or his family, how much more important is it to gain in the same way a little evidence regarding a prospective union which may prevent the transmission of insanity to generations. Other facts of the non-medical variety which should influence our decision are those relating to the surroundings of the future husband or wife. Is he or she likely to have a life of comfort and prosperity, or one of care, sacrifice and anxiety, or too great responsibility? Is there a great disparity in years? Will the neurotic companion be separated from congenial friends and surroundings? These points need only to be mentioned for us to recognize their importance in the question of the mental health of those in question.

We now take up the second class of difficulties, viz.: the medical problems to be solved in order to form as accurate an opinion as possible. These pertain to the etiology, forms and clinical history of mental disease, and concern both those persons who are predisposed to insanity by inheritance simply, and those who have in person experienced the disease. Afterwards are to be considered the dangers, viz.: those threatening the prospective mother, father or family and their property, perhaps through the appearance of insanity in either parent or both, or its transmission through inheritance to their offspring; and on the

other hand, those affecting the man or woman deprived of marriage through wrong advice.

The amount of hereditary taint through mental defect which should preclude marriage cannot be gauged with any approach to accuracy, as at present, in spite of diligent research, but a few general and elementary laws regarding the transmission of even the normal physical and mental condition in man have been ascertained so as to admit of accurate application to individuals. As every one knows, however, a certain class of physical diseases are more liable than others to reappear in descendants. So, in the domain of insanity, there are certain disorders that bear the hereditary stamp and are, therefore, more productive than others of harm to the offspring. In estimating the probabilities after ascertaining the *number* of ancestors who have been mentally deranged, the nature of their maladies is, therefore, of importance. Where the reappearance of insanity has long been a feature of the family history, the chances are evidently more unfavorable than if one or two immediate ancestors have had sporadic attacks of that disorder. Periodical insanity, dipsomania, epilepsy, imbecility, etc., are some of the prominent hereditary forms that are especially dangerous under these circumstances. Family peculiarities, defects, and diseases of the body have also a certain influence, such as poor physique, actual deformity, and especially frequent cases of phthisis in the ancestry. The size of families may furnish a little corroborative evidence as well as the temperaments of individual members. Another point to be considered is whether a parent's insanity has occurred before or after the birth of the person who is the object of consultation. Cases occur also where the insane ancestor is a woman, her insanity solely of puerperal origin, and the inquirer a man. Here, the chances are largely in favor of marriage. If general paralysis of the insane alone figures in the ancestry, immediate or remote, it is usually quite safe to disregard it, except in the case of a child born during the disease. This disorder does not spring from the soil of insanity proper, nor does it give rise to it, but is a cerebral malady and engenders cerebral dis-

orders. Prof. Ball and Regis, after a most thorough and instructive analysis based on extensive statistics collected by them, go so far as to affirm that the physician may say with perfect confidence that a child of a general paralytic by the fact of its being an offspring of a general paralytic, is in no way likely to become insane; that it has only to fear cerebral disorders by predisposition, and that, consequently, the two critical periods of its life are childhood, by reason of the danger of brain troubles at this period, and mature life the season of cerebral paralyzes and general paralysis itself. Apart from the liability to such accidents, the descendancy is absolutely normal. It might be added that the liability of a repetition of general paralysis itself in the offspring, except where it is begotten during the disease, is lessened by the fact that it could be propagated only through a predisposition, a tendency to cerebral excitement and not by the inherited effects of the disorder itself, which, contrary to the progress of ordinary insanity, is invariably fatal. It is well to keep in mind also that insanity is much more likely to be transmitted through the mother than the father, as it has been found that there is a much larger proportion of mothers than fathers among the insane, not to speak of the fact that, as Spitzka puts it, the relative preponderance of maternal influence in hereditary transmission is almost a dogma of natural history. The same author quotes Richarz as establishing the following order of liability to insane inheritance.

Mother insane, 1, the daughter resembling mother.					
"	"	2,	"	son	"
"	"	3,	"	daughter	" father.
"	"	4,	"	son	"
Father,	"	5,	"	"	"
"	"	6,	"	daughter	"
"	"	7,	"	"	" mother.
"	"	8,	"	son	"

Above all is to be considered the great importance of the mental and physical organization of the person himself. Has he the insane or neurotic diathesis; whether it be hereditary or acquired? If so, he should be dissuaded

from marrying if possible. The signs of such a constitution relate both to physical and mental anomalies (not to be enumerated here), both of birth and development, expressed it may be, in the cranial conformation on the one side, or an excitable, nervous, melancholic temperament, perhaps an exaggerated egotism on the other, or both together. A tendency to recurrent headaches and other similar signs of the neurotic diathesis call for consideration as corroborative evidence. Our examination, moreover, should not be confined to the person consulting us, as not unfrequently a personal or family history of disease is thus discovered in the other partner which is sufficiently marked to change a heretofore lenient view regarding the marriage or to confirm an unfavorable one.² If the parties are near of kin to each other, a very slight mental taint is sufficient ground for cautioning them against marriage.

But the thoughtful persons of this class who are sufficiently concerned by the possibility of the transmission of insanity to their children to ask our advice before marrying, are very few compared with those who have themselves experienced that disease. To such cases, all that has been said regarding the dangers from marriage in those who are simply predisposed to mental disease through inheritance naturally applies with much greater force. Where each party is known to have had an attack of insanity, the families of such persons should be immediately warned of the alarming consequences of such a marriage. When but one of the contracting parties has had several attacks, no matter what the form of insanity, recurrence has become a feature, and marriage is also to be strongly discouraged. Yet we have known, in more than one case, physicians of high rank in the profession and unquestioned ability to give qualified assent to the union of patients, men with histories of periodical mania, in one of whom the attacks were of the most extreme homicidal and destructive nature. The apparently

² Perhaps the case alluded to by Drs. Bucknill and Tuke (*Psychological Medicine*, p. 65), might have had a different ending had this precaution been taken. It was that of an individual, not who had once been insane, but the one who had risked having an insane partner for life who was the one to become the subject of mental disease.

sane interval in many of these cases is often marked by a certain amount of mental deterioration. In others, however, especially when the attacks are of very short duration, the return to mental health is quite complete, reawakening in the patient's friends the strongest hopes, and blinding their belief in further insanity or the probability of its transmission. Unfortunately, this turn of affairs may also lead to the hasty medical verdict.

When there has been but a single attack on either side, there is still cause for much hesitancy in sanctioning marriage. Many elements will enter into the decision besides those already mentioned. Nevertheless, there are not a few such cases in which marriage is justifiable and celibacy, with destroyed hopes and affections, a positive harm. Every specialist in mental disease meets with such instances, and however strongly impressed he may be, in the mass of cases, with the dangers that are so often unseen and disregarded, he has also to keep in mind that the minority who may safely marry, on the other hand, can only obtain their rights at his hands. The policy of the protection of the family and society through the individual is a wise one only when it leaves room for intelligent scientific discrimination. The chances of the reappearance of insanity in the person, and its transmission to the offspring, are greater if the mother has been insane than if the father has been mentally affected. The cases where the marriages of women so situated is allowable are, consequently, more exceptional than are those of men. It is important, moreover, to inquire whether the woman has passed the menopause, as there are not a few instances in which marriage would be permissible, and possibly beneficial, where there is no danger of the transmission of the insanity of the parents, and in a restricted number, even although there be a prospect of its recurrence. The outcome as to recurrence and transmission depends upon the causation of the person's insanity and its course, and it is much less difficult in this class of cases to ascertain the nature of the antecedent mental disease than where it is a question of family history. If the insanity occurring long before, has been fully recovered from, was of

short duration, in a man without the hereditary taint or the insane diathesis, especially if the attack was mild in its nature, unattended with pronounced impulses or propensities, and due to accidental causes, or such as were temporary and have entirely disappeared without a probability of return, he presents the most suitable conditions for favorably influencing our decision, especially if his future external surroundings indicate increased comfort in life and he is to marry into a sound, fat, non-nervous stock. On the other hand, when the insanity has been one of those forms which are the expression of a continuous neurotic condition, *e. g.*, dipsomania, hysterical insanity, epileptic insanity, or paranoia, all of which may present remissions simulating recovery, there is no question as to the nature of our advice.

Another point we have also to consider is the best time for giving our advice. Only those who have had to deal with such questions can realize the temptation to give an early opinion, when the patient is rapidly convalescing, and one is being pressed on all sides for a decision. We have, nevertheless, to defer it in all cases, except in hopelessly chronic ones of long standing. Should a patient unexpectedly make a full recovery and remain well long afterwards, we may be obliged to confess that we have exaggerated the dangers, and to allow the union to take place, provided, of course, the favorable conditions above indicated be also present. Again, if a patient must be told that marriage is out of the question, by those whose decision will be actually final with him, *e. g.*, that of the betrothed acting on our advice, he will be only able to bear the shock of a separation without a relapse after a considerable time has elapsed and recovery has been fully established.

The interval that should elapse between an attack and marriage is exceedingly important, as, if a child be begot while a trace of insanity lingers in either parent, the result is almost certain to be offspring who are idiotic, imbecile, or weakminded. In this connection, much might be said were it germane to our subject on the duty of the asylum physician as to the scrutiny of visits of husbands and wives to patients.

The possible dangers from unwise marriages of the neurotic and insane can hardly be exaggerated. By an attack developing in the father or mother as feared, more than one fellow-being is now involved and new and dearer ties will have been made only to be broken. The wife in many cases becomes worn by care, ill-health, and anxiety on account of her husband's illness, and their circumstances, perhaps reduced, became through the increased expense for his care and his inability to longer support the family. Added to this is the dread as to the possible outcome for the children through the re-enacting by them and their children of this domestic drama. This is especially to be feared if the patient recover, and it is a very melancholy aspect of this question that, as Tuke points out, in proportion to the number of recoveries obtained, so there is the probability of a greater number of cases of insanity through hereditary transmission.³ Any further enumeration of the probable ills to health, life and property would be superfluous here. They are well known, and yet we are often tempted to withhold the facts or to understate them out of mistaken consideration for the feelings of an anxious lover or relative instead of distinctly and boldly explaining the situation. On the other hand, if from the tendency to generalize, which we have, perhaps, already too often alluded to, we refuse in favorable cases to consider the advisability of marriage on the ground that no risks of the sort should ever be taken, as much misery may be caused through the disappointment of those who had made up their minds to marry, as would be likely to occur from the development of disease in the progeny. By giving such sweeping advice we go no farther than many of the general public who, recognizing the fact, now scientifically established, that most insane never permanently recover, and that many have relapses, some with, fewer without mental deterioration in the intervals, lose sight of the exceptional cases where marriage is really advisable. We believe, on the contrary,

³ If the attack occur early in life, the dangers are by so many years greater, through the longer period of life thus given for recurrence of attack or continuance of the disease and the greater length of the child-bearing period.

that we should use the same careful discrimination and consideration of all the extenuating circumstances that every physician exercises in advising patients with physical disorders as to their future. The subject should not be left without reference to the possible cases where the physician will fail to find the necessary elements upon which to found his opinion. In this event he will refrain, as Fournier cautions us to do in similar situations in questions of syphilis and marriage, from announcing a verdict, as the physician is not bound to have an opinion. Often, as we have shown, we have to advise delay until further developments, either in the line of the previous history or personal mental improvement or failure makes our course plainer.

Finally, in spite of a seemingly accurate weighing of probabilities, we have to acknowledge that in the present obscurity of the pathology of insanity our most painstaking investigations may meet with disappointment. Attacks of insanity unexpectedly recur, and recoveries, to our surprise, become permanent. Our point is simply that mistakes should be reduced to the least minimum possible along lines such as have been here indicated.

LARYNGEAL AND PHARYNGEAL PARALYSES.

By F. H. BOSWORTH, M.D.,

PROFESSOR OF DISEASES OF THE THROAT IN THE BELLEVUE HOSPITAL MEDICAL COLLEGE, N. Y.

I ASK attention to a consideration of paralysis as affecting the muscular structures of the pharynx and larynx.

In order that I may bring forward the question whether we are not, in the large proportion, if not in all cases, to consider true neuropathic paralysis in these regions an evidence of a lesion of the medulla. Of course we exclude cases of myopathic paralyses such as follow diphtheria, as well as those cases of recurrent laryngeal paralysis dependent on pressure on the nerve trunk.

That classification of laryngeal paralyses which is based entirely on the anatomy of the organ, such as is given in Mackenzie's¹ earlier work and followed later by myself,² must be abandoned as failing to make any distinction between true paralysis and myopathic conditions, such as simple muscular weakness.

In this manner our interest in laryngeal paralysis becomes centered really in two conditions, viz., unilateral or bilateral paralysis of the abductor and recurrent laryngeal paralysis. The consideration of pharyngeal paralysis is added as contributing weight to the view that the true clinical significance of either form is in the suggestion that it is due in most instances to some lesion of the medulla.

While the whole motor innervation of the larynx is derived from the recurrent laryngeal nerve, with the exception of the crico-thyroid muscle, which in the present consideration may be discarded, we find this organ endowed with two apparently separate and distinct functions, viz.,

¹ Diseases of the Throat and Nose, N. Y., 1881.

² Laryngoscope, Hoarseness and Loss of Voice. Am. ed., Philadelphia, 1869.

that of phonation and respiration, the movements by which these functions are accomplished being directed apparently through the same nerve trunk, yet possessing an action each so entirely independent of the other that they afford a subject of study from a physiological point of view of no little interest, and notwithstanding the many and able contributions which have been made to literature on this subject, it seems to me there are many questions which still remain unsolved, especially those connected with paretic conditions of the respiratory function, namely, unilateral or bilateral paralysis of the abductor muscles. That this disease occurred before the days of laryngoscopy cannot be questioned, yet a diagnosis can only be established by a physical examination; hence, our recognition of the affection dates from comparatively recent times, and the disease still is an exceedingly rare one. Mackenzie, in his first edition reporting but a single case, while Ziemmsen³ had collated nine cases. Mackenzie⁴ in his second edition reports having observed sixteen cases, while in a study of the subject made by myself in 1881,⁵ thirty cases were collated. In the same year Semon⁶ made a valuable contribution to the subject based on an analysis of twenty-two cases. In addition to this, valuable contributions have been made by Krause,⁷ Eisenlohr,⁸ Sokaloff,⁹ B. Frankel,¹⁰ Jean,¹¹ Senator,¹² Biermer¹³ (as cited by Gottstein), Oppenheim,¹⁴ Ollivier d'Angers¹⁵ (as cited by Gottstein), Gerhardt¹⁶ (as

³ Cyclopedia of Prac. of Med., N. Y., 1876. vol. vii., p. 959.

⁴ Diseases of the Throat and Nose, Amer. ed., 1880, vol. i., p. 442.

⁵ N. Y. Med. Jour., Nov., 1880.

⁶ Arch. of Laryngology, vol. ii., p. 197.

⁷ Neurologische Centralblatt, 1885, p. 543. Berlin Klin. Woch., 1886, No. 20, p. 651. Journal of Laryngology, July, 1888.

⁸ Deut. Med. Woch., 1886, No. 21, p. 363. Arch. f. Psychiatrie, vol. ix., p. 1-45.

⁹ Deut. Arch. f. Klin. Med., vol. xli., p. 458.

¹⁰ Berlin. Klin. Woch., 1886, No. 40, p. 675.

¹¹ Progres Medicale, 1876, No. 20.

¹² Arch. f. Psychiatrie, vol. xi.

¹³ Die Krankheiten des Kehlkopfes. Leipzig und Wien, 1888, p. 305.

¹⁴ Berlin. Klin. Woch., 1886, No. 40, p. 675.

¹⁵ Op. cit., p. 309.

¹⁶ Op. cit., p. 310.

cited by Gottstein), Stephen Mackenzie¹⁷ (cited by Gottstein), and Saundby.¹⁸

There are three views which have been advanced to account for this curious disease :

- I. That it is due to a peripheral lesion ;
- II. That it is due to a morbid lesion in the continuity of nerve ; and
- III. That it is due to a central lesion.

I. As regards the peripheral lesion : Mackenzie¹⁹ in his earlier work states that the cause of the disease is generally cerebral, "but morbid influences which affect both pneumogastric or both recurrent nerves may give rise to it," while in his later work²⁰ he makes the suggestion that owing to the exposed situation of the abductor muscles on the posterior aspect of the larynx they are especially liable to become the seat of injury, which added to the fact of their ceaseless activity renders them vulnerable, and suggests that this fact may act in a causative relation to the disease, apparently abandoning the idea that the disease may be of cerebral origin. Following the same line of argument, Smith²¹ explains the increasing dyspnœa, which apparently occurred in his reported case by the suggestion that the progressive weakening of the posticus muscles resulted in a prolapse, as it were, of the posterior wall of the larynx. It may be interesting to note, in this connection, that Smith's case subsequently suffered an attack of cerebral hæmorrhage. Gowers,²² in an ingenious course of reasoning points out the fact that the abductor muscles are inserted into the artenoid cartilages at an acute angle, while in the adductors this insertion is at a right angle. Hence, pressure affecting the nerve-trunk results in a paralysis, first of the abductors and subsequently of adductor muscles. Krause, in his admirable series of articles on this subject,

¹⁷ Op. cit., p. 319.

¹⁸ Birmingham Med. Review, Dec , 1886.

¹⁹ Op. cit., p. 214.

²⁰ Op. cit., p. 437.

²¹ British Med. Journal, July 13th, 1878.

²² Lancet, June 5th, 1886, p. 1077.

seems to have reached the conclusion that the disease is due to a spasm of the adductor muscles, this spasm being due either to a central lesion or is the result of reflex irritation caused by a lesion of the superior laryngeal nerve. I think it is noticeable that when we attempt to explain the phenomena of bilateral paralysis by a diseased condition of the muscular structures themselves we are dealing largely with pure theory or speculation rather than the direct teaching of clinical observation. A close study of the cases on which this theory is based it seems to me hardly justifies the view that a simple local lesion explains all the phenomena of the disease. Thus, in Smith's case the cerebral hæmorrhage which subsequently occurred, points very emphatically to a pre-existing cerebral disease, which giving rise in the first instance to paralysis of the abductors subsequently developed an apoplectic seizure. Furthermore, I recall no well-authenticated case of recovery from bilateral paralysis of the abductors, whereas simple myopathic paresis is not usually regarded as necessarily incurable. Still further it should be stated that the onset of abductor paralysis is invariably sudden and without warning, whereas a muscular paresis in the nature of the case must be slowly progressive. Krause's curious theory of muscular spasm is open to the same objection, I take it, as that of myopathic paresis, and furthermore still leaves us somewhat in the dark. No one has, as yet, demonstrated what constitutes muscular spasm. Certainly, as regards laryngeal spasm, I am strongly of the opinion that this chapter of laryngology is still to be written by a wiser hand than has yet undertaken the subject.

II. As to a morbid condition of the continuity of the nerve as a cause of the disease. Pressure on the recurrent laryngeal nerve or other morbid conditions which may interrupt the nerve current has been a favorite theory by which the phenomena of abductor paralysis is explained. The advocates of this theory are immediately met with what seems to me an insurmountable objection. In a former article on this subject I made this statement. "As to the suggestion that the disease is due to pressure on the recurrent nerve, and that

this pressure so far discriminates between the nerve fibres as to destroy the conductivity of those fibres alone which are distributed to the abductor muscles; it seems to me that the assertion is utterly untenable; that this might happen on one side alone, and that a tumor pressing upon the trunk of the recurrent nerve might so far select its point of pressure as to paralyze the abductor muscles on that side is among the possibilities; that this should happen on both sides and to both recurrent nerve trunks would be one of the rarest of coincidences; that it should happen in a large series of cases is simply beyond the pale of possibility." In one of Semon's cases pressure on the recurrent laryngeal nerves seems to have resulted subsequently in bilateral paralysis of the abductors. Here is a clinical observation which, it would seem, is exceedingly difficult to harmonize with the above assertion. The investigations of Exner and Weinzeig,²³ however, enable us to explain this case of Semon's, which, by the way, I think is quite unique. Both these observers have shown a crossing of the nerves of the larynx, especially of the superior laryngeal, by which the muscles of one side receive innervation from the nerve trunk entering the opposite side, while Mandlestam²⁴ has demonstrated that some of the fibres of the adductor muscles receive innervation from the superior laryngeal nerve. Ziemmsen²⁵ makes the statement that "paralysis of individual branches of the recurrent nerves may arise from incomplete lesions of the trunk as the result of unequal pressure, or when the nerve fibres are affected in an unequal degree by degenerative changes." Semon²⁶ completely endorsing Ziemmsen's statement, goes further and asserts "there is a distinct proclivity of the abductor fibres to become affected in such cases either at an earlier period than the adductor fibres or even exclusively." This proclivity-theory, so ably advocated by Semon, had already been advanced by

²³ Monatschrift f. Ohrenheilkunde, Dec., 1884.

²⁴ Monatschrift f. Ohrenheilkunde, Dec., 1884.

²⁵ Op. cit., vol. vii., p. 948.

²⁶ Loc cit.

Rosenbach²⁷ and based on the observation of a single case, and subsequently sustained at the London Congress.²⁸ I find it difficult to see wherein this idea of Semon's adds to our previous knowledge of the disease. What does he mean by proclivity? What is the essential anatomical or histological condition wherein lies this proclivity or constitutes a proclivity? It seems not unfair to suggest an analogy between this and that definition of the action of opium as producing sleep by its hypnotic powers. Moreover, if Semon endorses Ziemmsen's teaching that the individual laryngeal muscles may be paralyzed by unequal pressure on the nerve trunk or differentiated degenerative changes, we need seek no further for an explanation of the fact of the paralysis. The proclivity-theory becomes entirely superfluous and uncalled-for, if it is possible that the individual fibre of the nerve trunk which supplies the posticus muscles has been sought out and destroyed by any morbid process. I have no desire to ignore the teaching of the admirable series of experiments performed by Semon in connection with Victor Horsey,²⁹ although I do not think they sustain Semon's proclivity-theory. Briefly, these experiments were as follows: The recurrent laryngeal nerve in one of the lower animals was exposed and ligatured. At the end of several days the animal was killed, and examination showed degeneration of the abductor muscles, with but slight changes in the other muscles supplied by the nerve. In addition to this, another experiment was performed, by which the larynx was removed from the animal and the trunk of the recurrent nerve subjected to an electric current, demonstrating that the contraction ceased in the abductor muscles before the others, or, in other words, the abductors died first. The experiments of F. Donaldson, Jr.,³⁰ in connection with Prof. Martin in the laboratory of the Johns Hopkins University, differed from the above in that they were performed upon a living animal, although

²⁷ Breslau Zeitschrift, 1880, Nos. 2 and 3

²⁸ International Med. Congress—Trans., vol. iii., p. 222.

²⁹ Brit. Med. Journal, 1886, Aug. 28th and Sept. 4th, pp. 405 and 445.

³⁰ Trans. of the Amer. Laryngological Assoc., 1886 and 1887.

subsequently verified on the dead subject. The conclusions were virtually the same, in showing that as the result of stimulation of the recurrent laryngeal nerve by the electric current the adductor muscles survived the abductors. The admirable series of experiments made by Hooper,³¹ in connection with Prof. Bowditch, in the physiological laboratory of Harvard University, in the same line of investigation failed to substantiate the conclusions arrived at by Semon, Horseley and Donaldson, in that under electrical stimulus the abductor fibres showed an amount of vitality fully equal to the adductors. The fact, however, seems to be established beyond dispute that under stimulation the adductor fibres show greater vitality than the abductors, or, in other words, that the abductor fibres show a proclivity, if you like, to paralysis, Hooper's experiments alone failing to establish this view. This greater vitality of the adductor muscles is still further illustrated in those cases of bilateral ankylosis of the crico-arytenoid joint, improperly regarded as cases of bilateral paralysis of the abductor muscles. Thus in the two cases reported by Lefferts³² as the result of syphilis there was apparently bilateral paralysis. It seems to me, however, that a nicer diagnosis would show that the blood-poison had given rise to a local ankylosis of the crico-arytenoid joint and fixation of the cords in the median line due to the fact that the more powerful structure of the adductor muscles overcame the less resistance on the part of the abductors.

How far these experiments throw light on the causation of the abductor paralysis I think still remains an open question. That they prove a difference in the response to electrical stimulation, on the part of the phonatory and respiratory muscles of the larynx, is conceded, and I think we may naturally expect this would be the case. The two functions differ essentially in a very marked degree. The respiratory movements of the larynx are unceasing, commencing with the first breath of life and only ending with dissolution. The phonatory movements of the

³¹ Trans. Amer. Laryngological Assoc., 1885, '86, and '87.

³² N. Y. Med. Journal, Dec., 1878.

larynx, on the other hand, are intermittent. The respiratory movements are involuntary, the phonatory movements voluntary. These functions, therefore, necessarily involve certain differences in innervation. I think an analogy may be suggested between the posticus muscles and the cardiac muscles, in that their contraction never ceases during life. May we not venture a further analogy in that, under an electric stimulus, the heart is arrested in diastole, or, in other words, it is for the time paralyzed. Under the electric stimulus, if continued, the abductor muscle is paralyzed. The analogy, of course, is not complete, but a certain similarity in the action of these two muscles, the cardiac and the abductor, under the electric stimulus, is suggested.

After all, it is a question as to how far physiological experiment aids us in the elucidation of the finer points in neuropathology. Can the nice differentiation and delicate localization of a morbid process at the ganglionic centres, or in the continuity of a nerve, be in any way imitated, or even approached, by the harsh, coarse, and rude manipulations of the physiologist's scalpel and scissors? In clinical study, then, and analysis of symptoms as they present to us in the cases which come under our observation, I think we must seek for the true cause of these affections; and certainly, if my conclusions shall be correct, we will find a stronger confirmation here in the view that abductor paralysis is due not to a peripheral lesion or to disease of the nerve trunk, but to

III.—Some morbid condition of the nerve centres.

Mackenzie, in his later work, seems to have abandoned the idea that this disease was due to a central lesion, as suggested in his earlier work. In my article, already quoted, I wrote as follows: "Reasoning from analogy, considering the peculiar character of the respiratory movements of the larynx in that they are purely involuntary and also reflex, that the opening of the glottis constituting the respiratory movement is an independent action separate from all the other movements which take place in the larynx as the result of muscular contractions, it is fair to conclude that this function is presided over by an independent

ganglionic nerve centre, and that the disease in question consists in some degenerative change taking place in this portion of the nerve centres."

This article was written in 1880, and I think that such light as has been thrown on the subject since that time only serves to substantiate this teaching. Furthermore, as before suggested, our best information must come from the study of cases and analysis of their symptoms. The following cases, hitherto unpublished, are taken from my own note-books.

CASE I.—Bilateral paralysis of the abductors in the early stage of tabes.

H. S. W., aged 48, merchant, was referred to me, June 5th, 1883, by Dr. Crumm, of South Otslich, N. Y., with the following history. In October, 1881, as the result of exposure, he contracted a cold, resulting in sore throat and loss of voice. At the end of a week he had a violent attack of dyspnœa, lasting perhaps a minute, characterized with noisy inspiration. The sore throat and loss of voice disappeared, but during the next fifteen months he had almost daily recurrent attacks of inspiratory dyspnœa, lasting a few second at a time, with no other symptoms. In addition to this he had occasional attacks of neuralgia, with some impairment of general health, otherwise no change. When he consulted me, a chest examination revealed nothing abnormal, in the larynx the mucous membrane fairly healthy, while the vocal cords were nearly motionless and parallel in the median line. I gave an unfavorable opinion on the case, and sent him home. After leaving New York he gradually developed motor and sensory paralysis, beginning in the right lower extremity, then affecting the left, and ultimately the upper extremities. These symptoms began in the winter of 1883-4, a few months before his death, which occurred in the spring of 1885, with marked evidences of paralysis of the faucial and lingual muscles.

CASE II.—Unilateral paralysis of the palatal and pharyngeal muscles as the result of bulbar hæmorrhage.

H. L., German, aged 63, merchant, consulted me on the 19th of August with the following history. Three months

before, he arose in the morning with a feeling of general malaise, weariness on exertion, with a notable difficulty of articulation, and the feeling of an exceedingly thick tongue. On attempting to swallow at breakfast, the bolus lodged in his throat, giving rise to a threatened attack of suffocation until the mass was expelled. Fluids passed to the stomach slowly and with difficulty. The condition improved somewhat and until he consulted me, when he was able to take a fair amount of nourishment, although always with exceedingly great difficulty. An examination of the fauces showed the uvula slightly tilted to one side, while, on protrusion of the tongue, the tip was bent to the same side. His speech was slow and articulation thick. On pricking the pharynx and palate on one side, reflex contractions were excited with considerable difficulty, while on the other side the response was prompt. Nothing was noticeable in his movements in walking other than that they were slow and apparently feeble. On directing him to hop on one leg and then on the other, it was ascertained that it was impossible for him to stand or hop on the left leg. Treatment in this case was absolutely of no avail. The disease progressed slowly until the following spring, when he died of inanition. During the last three months of his life the only nourishment he received was administered through an œsophageal tube, deglutition being impossible. My diagnosis in this case was hæmorrhage into the medulla near the nucleus of the glosso-pharyngeal, pneumogastric, and hypoglossal nerves, with subsequent softening, invading the restiform and olivary bodies.

CASE III.—Paresis of the palatal and pharyngeal muscles as the result of obliterating endarteritis in a gouty patient.

H. M., aged 62, was a case similar to Case II., with the exception of the ultimate recovery. He consulted me, November 14th, 1886, with the history of rising in the morning a week previously with a feeling of general malaise and weakness of the limbs, which compelled him to lie down again. His speech was slow and thick, and articulation difficult. On his breakfast being brought to him in bed, he

found considerable difficulty in swallowing both solids and liquids, although he managed to pass the softer kinds of food into the stomach, while the liquids were regurgitated into the nose. An examination of this patient when I saw him, revealed the same symptoms as those of case No. II. The uvula was bent to the left, narrowing the palatal arch to that side, while the right arch was broader than normal. On protrusion of the tongue, the tip was turned to the right side. In attempting to stand or hop on one leg, a notable weakness of the left side was ascertained. This patient always enjoyed fairly good general health with the exception of occasional attacks of gout from which he had been a sufferer many years. This patient was under my care for two months, and made a fairly good recovery under the administration of general tonics, strichnia, and lithia, although even up to the present time there is a noticeable impairment in his articulation and lack of vigor in locomotion. The diagnosis in this case was obliterating endarteritis of one of the small vessels distributed in the medulla, undoubtedly the result of the gouty habit.

CASE IV.—Paralysis of the pharyngeal constrictors as the result of endarteritis deformans.

A. S. R., aged 52, merchant, consulted me, May 9th, 1888. Up to ten weeks before he consulted me he was perfectly well. On rising in the morning he found that he could not speak. There had been no promonitory symptoms, no giddiness, headaches, etc. On attempting to eat, he found that he could not swallow easily. Solid food seemed to stick in the throat and cause choking. There was slight weakness of the right hand, but no other paralytic symptoms of the extremities. He immediately began to be troubled with salivation, the tongue felt swollen, the lips could not be approximated to whistle or to say O. After this he went to Washington and Fortress Monroe, and seemed to improve under the warm weather. About a week before he consulted me he took cold, and the hoarseness, which had almost totally disappeared, and his difficulty in swallowing, which during this interval had not troubled him, returned. At the time of examination both

symptoms seemed ameliorated, but still present. Since the attack he had lost flesh and strength. There were no cardiac, renal, or pulmonary symptoms. On examination, the larynx was normal, no difference in sensation between the two sides of the palate; pupils sluggish to accommodation, react perfectly to light. No loss of power in the extremities, tendon reflex normal; no facial paralysis, no deviation of the tongue; no intrathoracic trouble found on physical examination. Retinæ normal, field of vision not limited. There was sluggish movement of the palatal and pharyngeal muscles on both sides. This patient was put under the administration of strichnine, and directed to return to Richmond and Washington for two months and abandon any idea of attending to business. At the end of this time I saw him again, and discovered a very satisfactory state of improvement, although there was still evidence of mild faucial paresis. This improvement has slowly continued, and he has assumed charge of his business in Maine. My diagnosis in this case was deforming endarteritis, probably the result of a gouty habit, there being no reason to suspect syphilis. The prognosis given was a guarded one, and made dependent on the careful regulation of his mode of life in the future.

The above cases are reported as containing many points of interest and offering much of suggestion in demonstrating the localized manifestations of the symptoms which arise from organic disease of the medulla. The next case is of special interest as illustrating how a localized morbid process in the medulla may give rise to both a laryngeal and pharyngeal paralysis.

CASE V.—Mrs. B., aged 53, consulted me, June 1st, 1888, with the following history. Several weeks ago she awakened somewhat suddenly in the morning with a violent cough, with difficulty in breathing, the difficulty being of an inspiratory character. In connection with this, there was moderate hoarseness and difficulty in swallowing. The dyspnœic attacks recurred daily, lasting a few seconds to a minute, and always characterized by a peculiar crowing inspiratory sound. These symptoms persisted without much

change until I saw her, the dysphagia being of such a character as to seriously impair general nutrition. The hoarseness improved somewhat, but there was a noticeable impairment in phonation; the voice was weak. When I examined her I found the vocal cord of the left side motionless in the median line, indicating complete abductor paralysis; while on the right side there was a very limited amount of motion, abduction being but partially accomplished. An examination of the fauces showed sensation normal, but a very sluggish reflex action on both sides in responding to impact of the probe, more marked on the left side. Speech slow and somewhat difficult, not scanning. No deviation of the tongue. Stands easily with the eyes closed; touches the tip of the nose and ears with the eyes closed. No paresis of the muscles of the extremities. Tendon reflex normal. There was a suspicion of syphilis in this case which indicated the administration of iodide of potassium. The result of treatment was in every way satisfactory. At the end of a month the cough, dyspnœa, and dysphagia had completely disappeared, although an examination of the larynx showed the left vocal cord still motionless, with an improvement of the movements of the right cord, while the faucial muscles also showed an equal degree of improved action. With the exception of a slight relapse in the following week, the further history of this case was one of improvement, although on the last examination there was still complete left abductor paralysis.

CASE VI.—J. H., aged 33, saloon-keeper, was referred to me by a Dr. Highland, of Amsterdam, N. Y., on October 3d, 1887, with the following history. Fifteen months ago he awakened in the morning with almost complete loss of voice, with no notable symptoms other than some rather obscure neuralgic pains with difficulty or sluggishness of movement in his left arm. These symptoms persisted for a few weeks and gradually disappeared, involving no notable discomfort or interference with his ordinary occupation. One year later he was suddenly seized with an attack of what he describes as choking, a sudden catching for his breath, followed by loss of consciousness which lasted an

hour. On emerging from this state, he was conscious of no new symptoms. His weak voice persisted and was perhaps a little more noticeable. At the suggestion of Dr. Hyland he consulted me on a casual visit to the city, at which time an examination revealed complete paralysis of the left side of the larynx, the cord remaining motionless in a cadaveric position. A thorough physical examination failed to reveal any discoverable lesion of the thoracic organs and no impairment in the organs of locomotion or speech. In this case there was probably an embolic formation in a small blood-vessel in his earlier attack, with a similar formation later, shutting off the blood-supply from a larger tract of the medulla. The interesting feature of this case lies in the fact that we have a bulbar lesion involving only the nuclei of those fibres of the accessory which go to make up the recurrent laryngeal nerve. That the lesion in this case was in the medulla I think can hardly be questioned in view of the fact of his second apoplectic seizure. This is notably substantiated also by many cases found in the literature of the subject, although I recall none reported in which the bulbar disease gave rise to recurrent laryngeal paralysis alone without involving other parts. Thus, in an interesting case reported by Hughling Jackson,³³ there was paralysis of the right side of the tongue with wasting paralysis of the right vocal cord, slight weakness of the limbs, especially on the right side. There was no post-mortem. The lesion was located by Dr. Jackson in the bulb. In another case by the same author,³⁴ there was sudden paralysis of the tongue on both sides, difficulty of deglutition, and loss of voice, with apparent paresis of the cords. There was no post-mortem. In this case, Dr. Jackson considered the diagnosis of bulbar disease as established. In another case,³⁵ there was paralysis of the seventh nerve, of the left half of the tongue and palate, and of the left vocal cord. Post-mortem examination showed softening of the medulla in the region of the anterior pyramid and nodes of the cere-

³³ London Hospital Reports, vol. i., 1864, p. 361.

³⁴ *Op. cit.*, p. 368.

³⁵ London Hospital Reports, vol. iv., 1867, p. 314.

bral arteries. There was a history of syphilis in this case. In another case of the same author,³⁶ there was paralysis of the sixth, seventh, and eighth nerves of one side, the right cord being in the cadaveric position. Post-mortem examination revealed extensive disease in the right side of the medulla and pons. There was also a history of syphilis in this case. In the same connection, a case is reported by Proust, as cited by Hallopau,³⁷ of a female, aged 68, who suffered from complete paralysis of the vocal cords and dysphagia, intelligence not being affected. The autopsy showed thrombosis of the left vertebral artery, obliteration of the posterior inferior cerebellar artery, and softening of the floor of the fourth ventricle in the region of the nucleus of the hypoglossal, accessory, and facial nerves. In a case reported by Senator,³⁸ there was difficulty of swallowing, sensory paralysis, impaired voice with nasal intonation. Laryngoscopical examination showed double recurrent paralysis of the vocal cords. The autopsy showed an area of softening involving the olivary body, nucleus of the hypoglossus, and a portion of the fibres of the vagus. In a case reported by Smith,³⁹ there was inspiratory dyspnœa. Laryngoscopy showed double abductor paralysis, the patient a short time after having an apoplectic attack with loss of consciousness and slight impairment of power in the upper and lower extremities of the left side. Smith attributes the laryngeal symptom to exposure to cold and over-exertion; but from the subsequent history of apoplexy, it seems quite possible that the laryngeal symptoms may have been due to a bulbar hæmorrhage. Biermier, as cited by Gottstein,⁴⁰ reports a patient, aged 56, who suffered from recurrent paralysis of the right side, together with paresis of the right half of the palate and analgesia of the mucous membrane of the right side of the mouth and of the integument about the right eye. The attack came on suddenly and was attended

³⁶ *Op. cit.*, p. 318.

³⁷ *Des Paralyses bulbaires*, Paris, 1875, History 23.

³⁸ *Arch. f. Psychiatrie*, vol. xi.

³⁹ *Loc. cit.*

⁴⁰ *Op. cit.*, p. 305.

by headache. The diagnosis was thrombosis of the posterior cerebral artery with a softening of the medulla. There was a history of syphilis. Dumeneil⁴¹ has written an elaborate article on unilateral paralysis of the soft palate, citing twelve cases in which he observed this symptom.

Case eight is worthy of note in that an autopsy was performed, the patient dying of cancer of the pylorus, which revealed an area of softening in the right restiform body about the size of a pea. In this case the paralysis was confined to the palate alone. Eisenlohr⁴² refers to three cases which came under his observation, showing a paretic condition of the cords and impaired voice. In one case there was thrombosis of the left vertebral artery and degeneration of the left pyramid and of the right to a certain extent, with sclerosis of the ependyma of the floor of the fourth ventricle. The second case showed atheromatous degeneration of the arteries of the base, narrowing of the vertebral, obliteration of the small vessels running from the basilar arteries to the pons, and degeneration of both pyramids, especially of the left. In the third case, a male patient, aged thirty-three, suffered from complete recurrent paralysis of the left side of the larynx, paralysis of the left side of the palate and left side of the pharynx, voice not affected. There was diminution of sensibility of the skin over the left trigeminus distribution the facial, motor portion of the fifth, and hypo-glossal nerves were intact. The post-mortem showed the left recurrent involved in a thickened pleura and degeneration of the bulb and upper part of the cord. Sokaloff⁴³ cites the case of a woman, thirty-five years of age, suffering from weakness of the right arm and leg, defective hearing, face drawn to the left, difficulty in swallowing, deviation of the tongue and uvula to the right, and paralysis of the left vocal cord. Later in the disease there was dyspnoea. The autopsy showed a glioma involving the left side of the pons, medulla and olivary body. Ollivier d'Angers⁴⁴ cites the case of a

⁴¹ Arch. Generales de Medicine, 1875.

⁴² Deut. Med. Woch., 1886, p. 363.

⁴³ Loc. cit. ⁴⁴ Loc. cit.

male, sixty-eight years of age, suffering from bilateral paralysis of the abductors, dysphagia, spasmodic attacks of coughing, paraplegia and weakness of the arms. The autopsy revealed an aneurism of the basilar artery, compressing the olivary body and the nuclei of the glosso-pharyngeal, vagus and hypoglossal nerves. Gerhardt⁴⁶ cites a case of left posticus paralysis with carcinoma, compressing the medulla. A similar case is reported by McBride, as cited by Gottstein,⁴⁶ in which carcinoma at the base of the skull gave rise to paralytic symptoms involving the hypoglossal and the glosso-pharyngeal nerves of the left side, left abductor paralysis and complete anæsthesia of the left half of the larynx. A somewhat similar case is reported by Nothnagel,⁴⁷ in which an abscess originating in the petrous portion of the temporal bone gave rise to paralysis and atrophy of the left side of the palate, anæsthesia of the left half of the larynx, abductor paralysis of the left vocal cord, dysphagia and paresis over the distribution of the left facial, abducens and trigeminus. Krause⁴⁸ mentions one case of abductor paralysis necessitating tracheotomy. Death occurred apparently from suffocation. The autopsy showed a clot in medulla at the nucleus of the vagus and spinal accessory nerves.

Perhaps no chapter in neurology better illustrates the point which we desire to emphasize, namely, that a laryngeal paralysis should always call our attention to the fact of its probable source in the medulla, than the clinical history of locomotor ataxia or tabes dorsalis. Duchenne, Fereol,⁴⁹ and Charcot⁵⁰ lay special emphasis on laryngeal crises, or cough and dyspnœa, as not infrequently accompanying the early stages of the disease, although not defining the specific condition observed on laryngeal examination. This point was still further and very fully elabor-

⁴⁵ Op. cit. ⁴⁶ Op. cit., p. 311.

⁴⁷ Wien. Mediz. Blatter, 1884, No. 9.

⁴⁸ Neurologische Centralblatt, 1885, p. 543.

⁴⁹ Gaz. Hebdomadaire, 1869, No. 7, p. 108.

⁵⁰ Progres Medicale, 1879, No. 17, p. 319.

ated by Cherschevsky,⁵¹ who still failed to describe definitely the laryngeal conditions. That the special condition should be so long overlooked is perhaps not remarkable when we consider, as Gottstein⁵² suggests, that unilateral paralysis of the abductors may give rise to no special symptoms with reference to the voice.

Krause,⁵³ in reporting three cases of *tabes*, gives a definite description of the laryngeal conditions as revealed by examination. In one of these cases there was anæsthesia of the lining membrane of the larynx and loss of reflex irritability. In the other two reflex was retained. In all these cases the cords were paralyzed in the median line. Krause explains this condition on the theory of spasm. As before stated, this view I regard as untenable, the condition being one of genuine paralysis of the abductor muscles, as illustrated in the case reported by Ross,⁵⁴ in which the characteristic symptoms of *tabes* were present, and an autopsy revealed degeneration of the medulla in the region of the columns of Goll and in the posterior nuclear zone and in the cerebellar crura. In the case reported by Kehler,⁵⁵ there was difficulty in swallowing, the right vocal cord was in the cadaveric position, the left was normal, there were also attacks of coughing, pharyngeal paralysis, and paralysis of the closers of the epiglottis. The autopsy showed ependymitis of the floor of the fourth ventricle, with sclerosis involving the vagus nucleus, especially the right. Oppenheim⁵⁶ cites a case of paralysis of the right side of the palate, right vocal cord, the ocular muscles and paræsthesia of the right lower extremities with loss of knee-jerk. Electrical stimulation of the right recurrent nerve in the neck did not cause abduction of the vocal cord, although a similar experiment performed on the left side was successful. This experiment would seem to indicate degeneration in the trunk of the nerve, and yet this would not necessarily contra-indicate disease of the medulla. No autopsy was made in this case. Jean⁵⁷ cites

⁵¹ *Revue de Med.*, 1881, No. 1.

⁵² *Op. cit.*, p. 323.

⁵³ *Berlin. Klin. Woch.*, 1886, No. 20, p. 651.

⁵⁴ *Brain*, London, 1888.

⁵⁵ *Zeitsch. f. Heilkunde*, 1881, p. 440.

⁵⁶ *Loc. cit.*

⁵⁷ *Loc. cit.*

a curious case of laryngeal cough with spasm of the pharyngeal muscles, the autopsy showing atrophy of the restiform body and posterior pyramids, atrophy of the filaments of the left pneumogastric and spinal accessory at their origin. Wegner⁵⁸ is quoted as reporting two cases of his own and making a compilation of other cases of tabes with laryngeal paralysis, in all of which the laryngoscopic lesion was the same, namely, paralysis of the abductor muscles. In a case reported by Semon,⁵⁶ of bilateral paralysis of the abductors, the patient developed well-marked symptoms of tabes two years later. Semon takes the ground that there is no necessary connection between the two, and attributes the paralysis to other causes. In the light of later reports this position is hardly tenable, laryngeal paralysis marking more probably the early development of spinal disease. Thus, in the first of my own cases the tabes did not develop for more than two years after the laryngeal symptoms set in. Saundby⁶⁰ reports a case of tabes in which there was paralysis of the abductors and paresis of the adductors with abnormal frequency of the heart's action. On autopsy, there was degeneration of the posterior fibres of the cord extending into the medulla, involving the posterior pyramids and restiform bodies. The recurrent nerve was in a condition of chronic interstitial neuritis.

We find thus in examining the literature of this subject from a purely clinical point, how strong the evidence is that a very large portion of cases of laryngeal paralysis have their origin in the medulla oblongata. Experience teaches us that the physiology of the brain is best studied through an investigation of pathological conditions. Certainly as far as the question of localization of function is concerned, by far the most valuable part of our knowledge of this subject is derived from a study of pathological conditions.

The question suggests itself in this connection then, as to how far we are justified in looking for a motor centre of

⁵⁸ *Annual of the Universal Med. Sciences*, vol. i., p. 89.

⁵⁹ *Clinical Society Trans.*, vol. xi., p. 141.

⁶⁰ *Birmingham Med. Review*, Dec., 1886.

the larynx in the cortical substance of the brain. In none of the cases above reported was there discovered any evidence of a cortical lesion, and yet we find the various muscular movements of the larynx, as well as sensation, completely abolished by morbid conditions confined entirely to the medullary tissues.

I think if one studies closely the case reported by Delavan⁶¹ and compare it with many of the cases above alluded to, the opinion certainly is warranted that Delavan's case was a bulbar clot rather than infusion into the brain substance proper. I can hardly justify his conclusion therefore that it establishes the existence of a motor centre of the larynx in the third frontal convolution of the brain. Krause,⁶² following the same line of investigation, made a series of experiments on lower animals, in which by irritating the surface of the brain at the base of the præfrontal convolution, he produced phenomena which would seem to lend weight to Delavan's theory, but when he resorted to more vigorous measures in destroying the brain tissue, his results were far from satisfactory. Masini⁶³ endorses this opinion of Krause without, however, substantiating his view by reporting any experimentation on his own part. I think in estimating the value of Krause's experiments it is necessary to bear in mind the statement already made of the very great difficulty of imitating the hidden processes of disease by the coarse and bungling methods of physiological investigation.

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⁶¹ N. Y. Med. Record, Feb. 14th, 1885, p. 178.

⁶² Arch. f. Anatomie und Physiologie. Phys. Abtheil, 1884.

⁶³ 12th Congress of Italian Physicians and Surgeons.

PHYSIOLOGICAL ACTION OF SULPHONAL.

By WM. F. SHICK, M.D.

(From the private Laboratory of Dr. Ott.)

SULPHONAL was discovered by Prof. Baumann in 1886, and its chemical formula is $(\text{CH}_3)_2\text{C}=(\text{SO}_2\text{C}_2\text{H}_5)_2$ (diethyl-sulphon-dimethyl-methane). It is a white crystalline powder, melts at 125.5° , and is soluble in from 18 to 20 parts of boiling water, recrystallizes on cooling, and is practically insoluble in water at the ordinary temperature, 450 parts, according to Kast, being necessary to dissolve it. Its solubility is much increased, however, by the addition of salts, hydrochloric acid, and peptones.

It is odorless and tasteless, except to the most sensitive, to whom it possesses a slightly bitter, unpleasant taste.

It was first clinically introduced by Prof. A. Kast,¹ of Freiburg, who speaks of it in the highest terms as an hypnotic. He says of it, "Sulphonal deserves a prominent position among medical agents, not only as an hypnotic for producing temporary insensibility, but for its property of assisting the normal periodical want of sleep. It produces no harmful effects on the elements of the blood, it does not attack and impair the stomach, and even in large doses it calls forth no dangerous derangement of the function of the heart or the vascular system." He found it especially useful in cases of nervous insomnia, where many of the more powerful or "drastic" hypnotics do much harm.

Since Kast's observations, many clinicians have given the drug a thorough trial, and their opinions in the main coincide with his. Dr. G. Rabbas² says: "Sulphonal in moderate doses of two to three grammes acts more safely and effectively than either amylehydrate or paraldehyde in even

¹ Berliner Klinische Wochenschrift, 1888.

² Berlinischer Klinische Wochenschrift, 1888, No. 17.

larger doses. Where patients are accustomed to the use of narcotics, sulphonal is even more successful." Moreover, he claims that the action is more prolonged than that of chloral, but not as prompt, sleep ensuing from one-half to two hours after the administration of the drug, lasting from six to eight hours. The sleep is normal and unbroken, leaving the patient very much benefited. He concludes by saying, "The results of my investigations induce me to give unqualified approval of sulphonal."

Dr. H. Rosin³ employed sulphonal in 274 separate cases, and says that he has found sulphonal, in doses of two grammes, is equally efficacious as morphia and chloral, and on account of its harmlessness he recommends it very highly. Dr. Carl Oestreicher has used it in over 100 cases, and speaks very highly of it. Dr. Julius Schwalbe reports its use in over 50 cases, and finds vertigo and headache as after-effects in a slight percentage of his cases. The ordinary dose of the drug is about thirty grains; and fifteen grain doses, according to Kast, are large enough for women. Forty-five grains have been given without unpleasant effect, though a drachm produced a deep sleep, lasting from eight to twelve hours, followed by a staggering feeling on the following day.

Rosin found thirty grains equal to one-sixth to one-quarter grains of morphia. Rabbas claims that doses of four grammes, alternated at short intervals with doses of two to three grammes, are harmless. Schmey⁴ found that two grammes in a case of angina pectoris produced no sleep, but aggravated the condition of the patient.

These observations induced me to study more carefully the physiological action of this drug. The article I used was sulphonal "Riedel." It was dissolved by the aid of warm water, and injected subcutaneously or by the jugular. The experiments were made upon frogs and rabbits.

GENERAL ACTION.

EXP. I.—A small frog received subcutaneously, at 3 P. M., 1.04 grains of sulphonal.

³ Berliner Klinische Wochenschrift, No. 25.

⁴ Centralblatt f. Medicinischen Wissenschaften, 1888, No. 46.

3.20 P.M.—Is very quiet.

3.25 “ All voluntary activity is abolished; reflexes unchanged.

4.35 “ In same condition as at 3.25 P.M. Next day was quite well.

EXP. II.—A large rabbit received at 3.25 P.M. Six grains of sulphonal in capsule, which was pushed down the œsophagus. At 5.00 P.M. no effect was observed, probably on account of its insolubility. The next day the animal was weak, staggered, and was inclined to be very quiet.

EXP. III.—Large rabbit.

2.40 P.M.—Received subcutaneous injections of 3 grains of sulphonal.

2.45 “ Pupil slightly contracted, and moves about.

3.15 “ 3 grains of sulphonal injected.

3.30 “ 9 grains of sulphonal injected.

3.35 “ Animal very quiet; when he attempts to move, staggers.

4.15 “ Animal asleep.

5.00 “ Animal deeply narcotised.

A cut was made in the ear, from which some blood was obtained; this was placed in the field of a Sorby-Browning spectroscope, but only the normal bands of oxyhæmoglobin were seen.

EXP. IV.—Rabbit. Normal pupil, $9\frac{1}{2}$ millimetres.

2.00 P.M.—Subcutaneous injection of 4 grains of sulphonal.

2.55 “ 4 grains of sulphonal injected; no change in the pupil.

3.00 “ Pupil 9 millimetres in diameter. Animal is very quiet; when made to move, staggers. When placed upon its side, does not attempt to right itself.

3.30 “ 18 grains of sulphonal injected subcutaneously. Pupil 8 millimetres in diameter.

4.00 “ Animal asleep.

Next day the animal is in a dazed and staggering condition.

These experiments prove that sulphonal by the stomach acts very slowly on account of its insolubility, that the sub-

cutaneous warm solution of sulphonal is much more active.

They also show that it is a narcotic, followed by a state of general relaxation of the muscles of the body and a staggering gait next day.

The oxygenation of the blood does not seem to be affected, using spectroscopic tests. The pupil is slightly contracted. Whether directly or indirectly due to the drug, I am not prepared to state.

MOTOR NERVES.

EXP. V.—Frog, unusually large, strong and active.

2.40 P.M.—Injected subcutaneously, 7 grains of sulphonal.

3.00 “ No loss of sensibility, quiet, weak in anterior extremities; when pinched, tried to hop, but was unable.

3.07 “ Injected .4 grains of sulphonal; struggles, lies flat on its back when placed in that position.

3.32 “ Injected .345 grains of sulphonal; sensibility diminished.

3.41 “ Animal is quiet, flattened out, no movement whatever, pupil slightly contracted.

4.00 “ Is lying on back, relaxed, unable to move; loss of all voluntary motion. Sciatic nerve irritable at 440 millimetres, Du Bois inductorium.

Several days later was in normal condition.

This experiment demonstrated that the motor nerves were quite irritable in deep narcosis.

MUSCULAR ACTION.

EXP. VI.—Frog received subcutaneously .6 grains of sulphonal; brain and spinal cord destroyed. The gastrocnemius was attached to Marey's myograph and loaded with a weight of ten grammes; single induction shocks were sent through the muscles, and the curves registered on the smoked drum of a Marey-Foucault regulator. The time of the contractions was noted by a tuning-fork registering 240 vibrations per second. Several experiments showed no change from that seen normally.

This led us to infer that the loss of sensibility is not due to lesions of the muscles.

SENSORY NERVES.

EXP. VII.—A small and lively frog had the blood-vessels of the right leg ligatured; .6 grains of sulphonal were injected. After a short time, the reflex action of each posterior extremity was tested by a watery solution acidulated with sulphuric acid. The time it took each extremity to be withdrawn was noted by the metronome making 100 beats per minute.

P.M.	LEFT LEG.	RIGHT LEG.
2.40	9	8
2.45	10	9
2.50	10	9
3.00	9	9
3.06	9	9
3.10	8	7
3.15	8	9
3.24	13	16
3.27	16	14
3.35	11	11

EXP. VIII.—Frog in the same condition as the preceding one; blood-vessels of the right leg ligatured at 3.40 P.M. Sulphonal given subcutaneously.

P.M.	LEFT LEG.	RIGHT LEG.
3.45	15	15
3.48	12	10
4.00	8	8
4.12	11	8
4.15	8	8
4.22	9	9
4.27	9	10
4.35	8	9

It is evident from the preceding experiments that as the loss of sensibility is equal in the leg in which the poison is excluded, the cause is not in the peripheral terminations of the sensory nerves. It has already been shown not to be in the muscles or motor nerves, hence it must be in the central nervous system.

REFLEX ACTION.

EXP. IX.—Frog. Cerebrum ablated. Reflex action tested by a metronome beating 100 per minute; irritant used, water slightly acidulated with sulphuric acid.

TIME.	METRONOME BEATS.
2.45 P.M.	27
3.12 "	20
3.15 " Injected .2 grains of sulphonol.	
3.16 "	31
3.20 "	49
3.35 "	33
3.43 "	58

EXP. X.—Conditions same as Exp. IX.

TIME.	METRONOME BEATS.
3.00 P.M.	20
3.08 " Injected .3 grains of sulphonol.	
3.24 "	25
3.27 "	39
3.40 "	55
3.45 "	44
3.49 "	34
3.56 "	38

EXP. XI.—Frog. Conditions same as in Exp. X.

TIME.	METRONOME BEATS.
2.30 P.M.	20
2.35 "	19
2.36 " Injected .6 grains of sulphonol.	
2.40 "	32
2.45 "	28
2.50 "	32
2.55 "	27
3.00 "	26
3.05 "	23
3.10 "	23
3.15 "	21

EXP. XII.—Frog. Conditions same as Exp. XI.

TIME.	METRONOME BEATS.
2.30 P.M.	15
2.35 "	14
2.37 " Injected .3 grains of sulphonol.	
2.45 "	10
2.55 "	4
3.00 "	4
3.05 "	4
3.10 "	3
3.15 "	3
3.35 "	3

EXP. XIII.—Conditions same as Exp. XII.

TIME.	METRONOME BEATS.
2.40 P.M.	20
2.43 "	20
2.45 " Injected .6 grains of	sulphonal.
2.55 "	
3.00 "	38
3.05 "	40
3.10 " Cut off the medulla.	46
3.15 "	20
3.20 "	15
3.25 "	16
3.30 "	23
3.40 "	30
3.45 "	32

EXP. XIV.—Conditions same as Exp. XIII.

TIME.	METRONOME BEATS.
3.00 P.M.	28
3.05 "	26
3.11 " Injected .6 grains of	sulphonal.
3.15 "	
3.20 "	25
3.25 "	28
3.35 "	30
3.45 "	27
3.50 "	28
3.55 "	18
4.00 " Injected .6 grains of	sulphonal.
4.04 "	
4.10 "	42
4.13 "	23
4.25 " No response.	25

EXP. XV.—Frog. Entire brain severed from the cord; animal unusually lively.

TIME.	METRONOME BEATS.
3.00 P.M.	20
3.05 " Injected .6 grains of	sulphonal.
3.10 "	
3.15 "	27
3.18 "	22
3.21 "	21
3.24 "	22
3.29 "	15
3.35 "	15
3.40 "	17
3.47 "	22

EXP. XVI.—Frog. Conditions same as Exp. XV.

TIME.	METRONOME BEATS.
2.45 P.M.	16
2.50 " Injected .2 grains of sulphonal.	
2.55 "	24
3.00 "	13
3.03 "	13
3.06 "	15
3.11 "	19

EXP. XVII.—Frog. Conditions same as Exp. XVI.

TIME.	METRONOME BEATS.
3.49 P.M.	13
3.57 " Injected .6 grains of sulphonal.	
4.01 "	12
4.06 "	12
4.11 "	10
4.16 "	15
4.20 "	12
4.25 "	14

EXP. XVIII.—Frog. Conditions same as Exp. XVII.

TIME.	METRONOME BEATS.
4.50 P.M.	16
4.52 " Injected .2 grains of sulphonal.	
4.56 "	14
5.00 "	16
5.05 "	24
5.10 "	25
5.15 "	21
5.18 "	22
5.25 "	21

Usually it reduces reflex action, occasionally it increases it.

When Setschenow's inhibitory centres are removed, sulphonal does not diminish the reflex activity. The inference would be that it stimulates Setschenow's centres, thus decreasing reflex activity.

CIRCULATION.

EXP. XIX.—Frog. Brain destroyed, heart exposed.

TIME.	HEARTBEATS PER MINUTE.	REMARKS.
2.45 P.M.	22	
2.50 "		Injected .8 grains of sulphonol.
2.55 "	28	
3.00 "	25	
3.05 "	25	
3.10 "	24	
3.15 "	22	
3.20 "	22	
3.25 "	21	
3.30 "	20	
3.45 "	19	
4.00 "	19	

EXP. XX.—Rabbit, etherized; carotid and jugular prepared, Ludwig's Kymographion attached.

TIME.	PULSE.	PRESSURE.	REMARKS.
3.40 P.M.	42	72	
3.40.15 "	46	76	Injected 2½ grs. sulphonol.
3.40.30 "	40	40	
3.41 "	41	64	
3.42 "	44	68	
3.44 "	44	76	
3.45 "	46	72	
4.00 "	46	88	
4.01.15 "	48	90	
4.01.30 "	45	68	Injected .5 grs. sulphonol.
4.01.45 "	46	48	
4.02 "	46	44	
4.03 "	44	74	
4.04.15 "	46	88	
4.21 "	44	110	
4.22 "	46	108	
4.23.15 "	44	100	
4.24.15 "	49	98	
4.24.45 "	46	102	

EXP. XXI.—Conditions same as Exp. XX.

TIME.	PULSE.	PRESSURE.	REMARKS.
3.32.15 P.M.	53	94	
3.32.30 "	55	86	Injected 3.7 grs. sulphonol.
3.32.85 "	59	82	
3.33 "	59	84	
3.34 "	59	98	
3.35 "	59	96	
3.51 "	58	116	
3.52.30 "	63	122	
3.54.30 "	62	122	

EXP. XXII.—Rabbit. Conditions same as in Exp. XXI.

TIME.	PULSE.	PRESSURE.	REMARKS.
2.37.45 P.M.	63	116	
2.38 “	63	118	Injected 2 grs. of sulphonol.
2.38.15 “	63	94	
2.38.30 “	63	94	
2.39 “	63	110	
2.41 “	62	122	
2.47 “	59	120	
2.47.30 “	61	118	
2.47.45 “	61	118	Injected 3.7 grs. sulphonol.
2.48 “	62	110	
2.48.15 “	64	106	
2.49 “	60	114	
2.50 “	58	122	
2.53 “	—	124	
3.04 “	—	116	
3.04.15 “	—	104	Injected 5.5 grs. sulphonol.
3.04.30 “	—	64	
3.05 “	—	—	Animal killed.

These experiments demonstrate that upon the heart sulphonol has but little activity, usually slightly increasing its rapidity. As to arterial tension, immediately after the injection by the jugular it is decreased; afterward, however, it is increased considerably; the opposite case happens with chloral.

RESPIRATION.

EXP. XXIII.—Rabbit. Tracheal canula attached to Marey's polygraph; jugular prepared.

TIME.	RESPIRATIONS.	REMARKS.
2.45 P.M.	31	
2.45.15 “	29	
2.45.30 “	29	
2.45.45 “	29	Injected .2 grains of sulphonol.
2.46 “	30	
2.46.15 “	25	
2.47 “	17	
2.48 “	16	
2.48.15 “	14	
2.48.30 “	13	
2.50 “	12	
2.51 “	11	
2.53 “	8	Animal killed.

EXP. XXIV.—Conditions same as *Exp. XXIII.*

TIME.	RESPIRATIONS.	REMARKS.
3.35 P. M.	14	
3.35.30 "	20	
3.36 "	20	Injected .1 grains of sulphonol.
3.36.15 "	21	
3.36.30 "	15	
3.37 "	13	
3.37.30 "	11	Pulse, 254.
3.45 "	10	
3.48.30 "	15	
3.48.45 "	13	Injected .1 grains of sulphonol.
3.49 "	16	
3.51.15 "	10	Pulse, 280.
3.58 "	12	
4.05 "	13	
4.05.30 "	15	Injected .2 grains of sulphonol.
4.05.45 "	11	
4.06.15 "	12	
4.06.30 "	12	Injected .2 grains of sulphonol.
4.07 "	13	
4.08.45 "	12	
4.09.45 "	14	
4.10 "	15	
4.11 "	14	
4.13 "	14	

Animal taken off, lively, no staggering.

EXP. XXV.—Rabbit. Vagi cut.

TIME.	RESPIRATIONS.	REMARKS.
4.30 P. M.	7	
4.30.15 "	8	
4.31 "	9	Injected .2 grains of sulphonol.
4.31.15 "	9	
4.31.30 "	10	
4.32.15 "	11	
4.32.30 "	12	
4.33 "	13	
4.34 "	12	
4.36 "	13	
4.37 "	12	
4.40 "	11	
4.46.45 "	12	
4.47 "	10	Injected .2 grains of sulphonol.
4.47.15 "	11	
4.47.30 "	8	
4.48 "	8	

TIME.	RESPIRATIONS.	REMARKS.
4.49 P.M.	8	
5.50 "	9	
4.50.30 "	10	
5.51.45 "	9	
5.00 "	9	
5.04 "	9	
5.05 "	8	
5.08 "	5	
5.09.15 "	7	
5.10 "	9	
5.11.15 "	9	

The preceding experiments prove that sulphonal decreases the rapidity of the respirations.

Section of the vagi does not alter the result.

My conclusions as to sulphonal are as follows :

(1) It does not affect the irritability of the motor nerves.

(2) It does not alter the muscle curve.

(3) The sensory nerves are left intact.

(4) It depresses reflex activity mainly by an action on Setschenow's centres, occasionally it exalts reflex excitability.

(5) It acts as a narcotic.

(6) The pulse is usually somewhat accelerated.

(7) The arterial tension, after a temporary fall, is considerably increased.

(8) Respiration is depressed, section of the vagi does not alter the effect.

These facts lead me to believe that sulphonal will replace chloral to a considerable extent. The well known dangerous action of chloral as to heart and respiration is avoided with this drug, and if the narcotic effects are equal sulphonal should have the preference. Whilst I have seen the heart paralyzed by the drug in a few minutes, yet it was due to the sudden action of the drug by the jugular and perhaps partly to some of the drug being thrown down on account of its insolubility, for the solution was somewhat warmer than the temperature of the blood.

ANEURISM OF AN ANOMALOUS ARTERY CAUS-
ING ANTERO-POSTERIOR DIVISION OF
THE CHIASM OF THE OPTIC NERVES
AND PRODUCING BITEMPORAL
HEMIANOPSIA.¹

By S WEIR MITCHELL, M.D.,

MEMBER OF THE NATIONAL ACADEMY OF SCIENCES.

NOTE-BOOK, Case No. 836, D. G. H., æt. 43, manufacturer of bromine, from the Springs in Kentucky, consulted me May 29th, 1886. He was a large, florid, wholesome looking man without previous grave disease. He had had for thirty years hæmorrhoids, which at times bled freely. All of his functions, digestive, secretive and reproductive, seem to have been perfect. Of late, he had felt fatigued from any unusual exertion; also, the legs and arms became easily numb in sleep, or from pressure or malposition when awake. He has had for a year varying but gradually increasing pain in the parietal and vertex regions, and at times this pain darts through to both temples or to either. Excessive exertion causes it to increase if present, or may bring it on.

To shake the head and jar it causes no pain.

Hearing good. At no date had he any noises in the head.

Vertigo none. Station normal, with eyes open or shut. Knee-jerk normal.

Olfaction natural; but not examined later in the case.

He says that three years ago, during very hot weather, he became abruptly weak in the legs, so that he fell on his knees and hands, but did not lose consciousness. For a few hours his right foot dragged, but he had been able to rise and walk, and had no other annoyance than great sense of fatigue.

¹ Read at the meeting of the American Neurological Association, Washington, D. C., September, 1888, by Dr. F. X. Dercum.

The following is the report of Professor William Thomson of the state of the eyes in Mr. H.'s case :

"On May 29th, 1885, when first examined, the patient stated that eighteen months ago he began to have trouble with his sight in the form of diminished vision in the left eye, particularly towards the left side, which gradually increased, and that six months later the right eye began to fail in vision towards the right side. The acuteness of vision is R. E. $\frac{6}{60}$, L. E. $\frac{6}{60}$, equals $\frac{1}{10}$, and at 20 centimetres, he reads D = 2, showing same acuteness. Has full power of accommodation, normal sensibility of each iris to light and perfect fixation of each eyeball and consequent integrity of each third nerve. Sensibility, taste, smell and hearing are normal. He has entire loss of sight to the right of the vertical line with the right eye, and to the left of the same vertical line with the left eye, showing complete anæsthesia of the nasal half of each retina. This was carefully determined by the use of the two lights of my ametrometer and was accurately vertical for the left eye, but inclined slightly in the right eye, being 10° to the right in the upper and 5° to the left in the lower part of the field. The color sense was yet good in the remaining half of each field, and he recognized promptly red and green when the lights were tinted with glasses of these colors.

By ophthalmoscopic examination the fundus presented no changes except at each papilla, where the vessels of the retina appeared perhaps somewhat attenuated, whilst the neuroglia, especially of the left eye, was pale, the porus opticus enlarged, and the appearances those of partial atrophy. There was no swelling of either papilla, nor any change in the retina that would indicate a previously "choked disc."

The diagnosis was pressure at or in front of the chiasm, sufficient to cut off the connection between the inner halves of each retina, with neuritis or partial atrophy, especially of the left nerve to account for the low acuity of vision, caused by some growth which did not excite enough meningitis to cause a swollen papilla.

On November 16th, 1885, the only change observed was an increase of acuteness in the right eye to $\frac{1}{3}$, being for the

R. E. $\frac{6}{18}$ and for the L. E. $\frac{6}{60}$, with more marked appearances of atrophy in the left papilla. The fields of vision were carefully determined by the perimeter, *vide* Figs. 1 and 2. He was found to have slight astigmatism in each eye, but this was disregarded, and I gave him $+1.5$ $\frac{\text{Rt.}}{\text{Lt.}}$ for his near vision, with which he could read with the right, D = 050 at 25 centimeters.

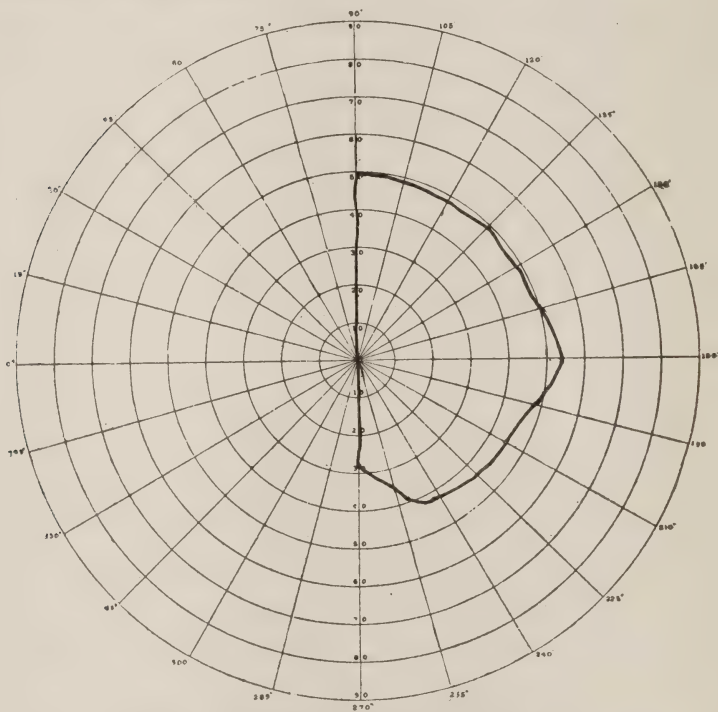


Fig. 1.—Illustrating Field of Left Eye.

June 16th, 1886, R. E. V. = $\frac{6}{18}$, L. E. $\frac{6}{60}$ by excentric fixation, the hemiopia being the same, but difficult now to determine on the left, except with lights.

Feb. 7th, 1887, R. E. V. = $\frac{6}{18}$, L. E. $\frac{1}{30}$ only; hemiopia as before, pupils responsive, dilated with cocaine, both papillæ more white, retinal vessels normal and full in size. Although the left was now reduced to $\frac{1}{30}$ only, the hemiopia was readily detected with the ametrometer."

In February, 1887, Professor Harrison Allen examined the nose, and found much discharge of thin mucus. He

thought that there must be disease of the mucous membranes of the larger sinuses.

The headaches continued, but did not increase. Once or twice he had a passing sense of mental confusion. At intervals of six or eight months, Mr. H. consulted me, and I saw him some six weeks before his death. There had been no remarkable addition of new symptoms, and the old ones were unaltered. Still later, May 11th, he was in my

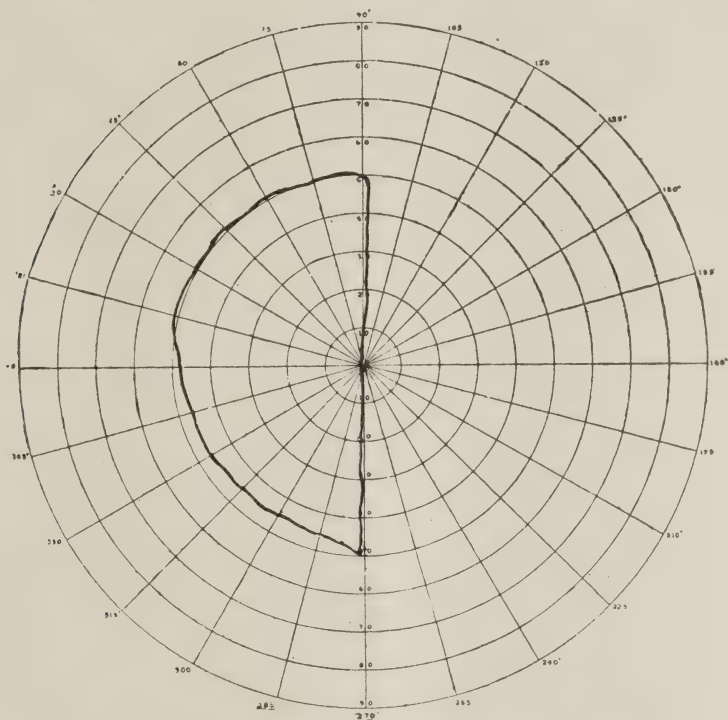


Fig. 2.—Illustrating Field of Right Eye.

consulting-room, he talked of bromine and its influence on the health of those who work in its manufacture. He was clear-headed and interesting. On May 20th he paused in Baltimore at the house of a relative. Complaining of headache, he was persuaded to remain over night. After 6 P. M., his pain being worse, he became sleepy, and in a few hours comatose. He died suddenly at 5 A. M., May 21st.

It so chanced that Dr. J. T. McLean, of New Philadelphia, Ohio, as well as his wife, who also is a physician,

were aware of the deep interest taken in this case by Professor Thomson and the author. They made, therefore, earnest efforts to secure an autopsy and at last succeeded. Without them the instructive lesson of this case had been lost. I append here the notes of the cadaveric section as made by Dr. McLean.

"Autopsy, May 24th, at 8 A. M., seventy-five hours after death: Rigor mortis slight. External appearance shows marked purple discoloration of both ears, with large purple blistered surface on back of neck. Otherwise appearance of one dying in apparent perfect health. Weight 200 pounds. Eyes: pupils widely dilated, with marked congestion of all conjunctival vessels, ocular and palpebral. All the vessels of the scalp and external surface of calvarium, markedly congested. Dura mater not adherent except on inner surface along each side of longitudinal sinus, at which points slight exudation of lymph was found. All vessels of the pia mater of a deep purple color and in a state of extreme congestion. Nearly fourteen ounces of serum escaped during removal of the brain.

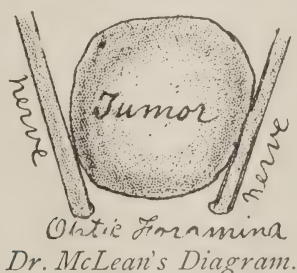
Elevating the anterior lobes of the cerebrum, the olfactory nerves and bulbs came into view and also the optic nerves entering the optic foramenia. The optic nerves were widely separated (fully one inch) by a large purple tumor filled with fluid, lying directly between them. About half an inch of each nerve could be traced backward from the optic foramen on each side of the tumor, which came up out of the sella Turcica and evidently by pressure had caused absorption of the olivary process and the optic groove as far as its extreme anterior border. A separation seemed to have taken place in the centre of the optic commissure, pushing the optic nerves and tracts to the outside of the tumor. The commissure could not be found. In attempting to dissect the tumor from the deep cavity in the sella Turcica, the upper rounded extremity slipped from its cavity in the brain, the centre of which cavity could be marked by a point directly in the centre of the circle of Willis. The right and left internal carotids were found

intimately connected with and apparently forming the tumor. They were divided by the knife at its upper border just before giving off the posterior communicating branches. The entire brain was now removed, leaving the tumor deeply imbedded in its cavity in the sella Turcica. The tumor was in no way attached to the brain except by the internal carotids, but was firmly attached to the bone around the border of the deep cavity which it had formed in the sella Turcica. The tumor being removed, the bony cavity was found quite rough and honey-combed. No further examination was made of tumor or brain, but both were immediately forwarded to Drs. Thomson and Mitchell.

Signed, J. T. McLEAN."

So anxious was I as to the accuracy of the examination that I asked in a letter, if the chiasm could possibly have been torn during the autopsy. To this query Dr. McLean replied as follows:

"The optic nerves were not torn. As the anterior lobes of the cerebrum were gently and gradually lifted, the olfactory nerves and bulbs being in place and perfect, the optic nerves came into view. By the aid of a rough dia-



gram, I perhaps can make it plain to you what I saw, viz., what seemed to be the neck of a large purple tumor coming up from the sella Turcica and pressing far forward directly between the anterior clinoid processes and directly between the optic foramina. The optic nerves were plain and distinct and perfectly straight on each side of the tumor, entering the optic foramina, and I noticed the peculiar direction, viz., widely separated and slightly converging, as here represented.

"Gently lifting anterior lobes in order to get more room to work, the large upper bulbous portion of the tumor slipped from the cavity in the cerebrum, as heretofore described. If it were possible for the commissure to have been pressed far behind the tumor, it might have been torn when the upper rounded part of the latter slipped out of the cavity it had formed in the cerebrum, but no trace of the commissure could be found, but only the two long straight optic nerves and tracts, as you no doubt observed in the specimen."

The specimen as it arrived was examined by the author with Professor Wm. Thomson and Dr. F. X. Dercum, and all were fully satisfied as to Dr. McLean's accuracy. The following study of the parts sent was made by Dr. Dercum, to whom is due also the collection of anomalies.

The case speaks for itself, as it stands alone in the records of pathological accidents. It seems needful to suppose that an anomalous artery connected the carotids by passing under the chiasm. This branch became aneurismal and enlarging, lifted the chiasm until this parted in the middle line, leaving a nerve on each side, thus dividing the right and left fibres which, crossing in the chiasm, supply the nasal sides—the temporal visual fields of each eye. At what date the final division took place it is impossible to say, nor does the ocular study made by Professor Thomson settle this point, since as early as the first examination of the eyes there was complete anæsthesia of both nasal halves of the two retinas. The left eye had evidently suffered most, and as time went on continued so to do. The absence of choking or optic neuro-retinitis is a notable fact, and that the presence of a pulsating mass as large as a lemon, caused so little disturbance of mind or of motor or sensory functions is interesting.

If any doubt remained as to the uses and functions of the internal bandelettes, this case assuredly settles the question in a most decisive fashion. It is a slowly done vivisection, effected with the least possible disturbance by a pathological process.

I leave any further discussion to my friend, Dr. Dercum.

When the brain was examined by Drs. Mitchell, Thomson and Dercum, it was floated in alcohol with its base upward. It was noted at once that the various structures normally occupying the space included by the crura and the basal portions of the frontal and temporal lobes were entirely wanting. No trace of an infundibulum or a chiasm was discoverable. In their stead, a huge cavity extending deeply into the brain was found. The walls of this cavity were irregular, softened, and disorganized. Lying to either side of it were revealed two white and rather ragged bands. These evidently corresponded to the optic nerves and tracts described by Dr. McLean in his record of the autopsy. Their anterior portions certainly looked like the optic nerves, but as they were followed backwards they became attenuated and ragged. In fact nothing but the anatomical relations of the posterior portions could justify one in calling them the optic tracts. The nerve on the right side, though imperfectly, was the best preserved; that on the left side was exceedingly difficult to follow, for, in the region of the destroyed chiasm, it was reduced to a few mere shreds. In addition, both nerves were so soft and brittle that the most careful handling caused them to break and tear.

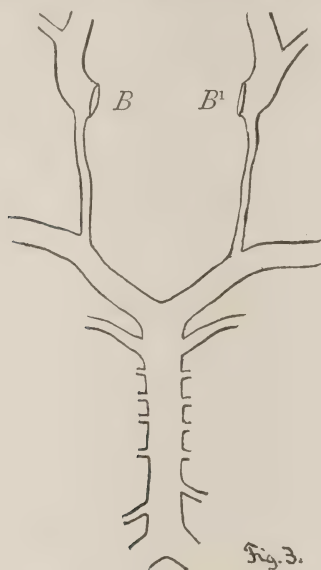
The circle of Willis was next carefully dissected. It is represented diagrammatically in the accompanying figure.

The openings B and B' communicated directly with the aneurism about to be described, *i. e.* judging from the relation of the parts and the accurate account of Dr. McLean. The tumor, though evidently shrunk by the alcohol, was decidedly larger than an egg and pyriform in shape. It had been ligated at the smaller end with a thread, and when originally examined was distended with fluid, and was of a dark, purple color. The larger portion, or base, fitted readily into the cavity of the brain already described; in other words, the larger end had been directed upwards, while the smaller end had occupied the sella Turcica. Upon handling a quantity of blood oozed out notwithstanding the ligature.

Careful dissection revealed two large openings at the smaller end. These communicated freely with the interior,

and were so directed that they faced slightly downwards and to the right and left respectively.

It requires no great effort of the imagination to conceive of these openings as originally confluent with the openings B and B¹ in Fig. 3. Indeed this explanation becomes more



probable when we reflect that the condition of the parts admits of no other. No other opening was detected in the cavernous carotids, and no other anomalous vessels were present in the circle of Willis from which the aneurism could arise.

A difficulty, notwithstanding, suggests itself. What has become of the remaining or pre-circular portion of the encephalic carotids? Naturally we should expect it to be adherent either to the circular portion of the carotid or, if severed from the latter, adherent to the aneurism. Evidently and very naturally, Dr. McLean in removing the tumor practiced two incisions. First, he separated the tumor from the carotids at a point "just before giving off the posterior communicating arteries." The doctor further tells us that, on removing the brain after this division, the tumor remained "deeply imbedded in its cavity in the sella Tur-

cica," and also that "the tumor was in no way attached to the brain except by the internal carotids, but was firmly

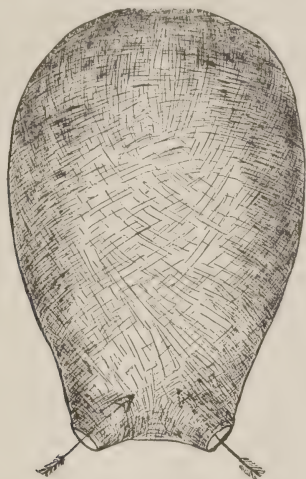


Fig. 4.

attached to the bone around the border of the deep cavity which it had formed in the sella Turcica." He therefore practiced a second incision in the final removal of the

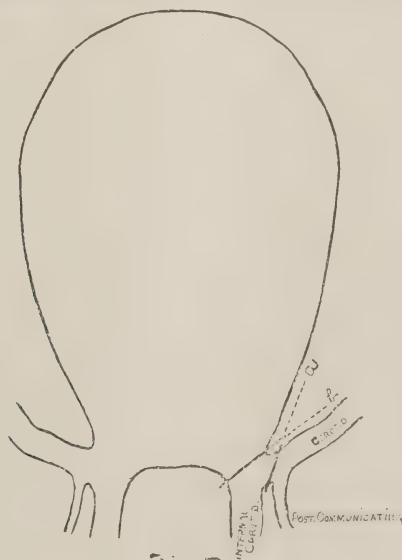


Fig. 5.

tumor, thus severing it from the pre-circular carotid. See Fig. 5, which shows the probable relation of the parts and the direction of the incisions. Line *a* represents the direction of the first cut, following which the brain together with the circle of Willis was removed, leaving the tumor still in the cranial cavity. Line *b* represents the cut which finally separated the tumor from the remaining portion of the carotid.

An attempt was made to study the optic nerves and tracts and the two cunei microscopically; but owing to the softened condition of the structures, the attempt was not successful. The parts had been macerated during the journey in an insufficient amount of alcohol, and post-mortem changes had evidently taken place.

The remnants of the nerves and tracts revealed nothing definite, except perhaps a relative excess of connective tissue elements. The cunei also showed no decided evidences of pathological changes, except perhaps the left. The latter showed here and there marked dilatation of the pericellular lymph spaces; yet whether these appearances indicated cystic degeneration, or were merely the outcome of a stronger alcohol acting upon an already softened cortex, is problematical.

In order to throw additional light upon this remarkable case, a collection of anomalies of the circle of Willis was made, excluding of course abnormalities of the basilar and pre-circular portions of the carotids.² This study has not proved without benefit. In explaining this case, Dr. Mitchell assumes the existence of an anomalous vessel connecting the two carotids. That such a vessel did exist, there can I think be no doubt. In fact, that there is a general tendency to the anastomosis of the opposite sides of the circle of Willis is shown by the great constancy of the normal anterior communicating artery as well as by its anomalies. Regarding the latter, there may be many varieties. We may have, for instance, instead of one vessel, two. Incoronato and Barbieri record such cases, and Spitzka has given me the notes of a third. These instances of double anterior com-

² See Appendix.

municating artery are very significant. They present, as it were, an exaggerated expression of the normal tendency to anastomosis. Again, it surely makes but little difference in the functional value of the anterior communicating artery just at what point it crosses the circle, whether it connects the anterior cerebrals high up in their course or low down. After all, it is only a question of the relative position of the transverse vessel as to whether it can influence by aneurismal disease the optic chiasm. If it be sufficiently low down, interference with the chiasm becomes inevitable.

In order to show that Dr. Mitchell commits no breach of anatomical probability in supposing an anomaly to have existed, it may be stated that almost every conceivable variation has been known to occur. Thus, to begin with, the anterior communicating artery may be entirely wanting. Barbieri and Spitzka have each reported such cases. Again, the vessel may be present, but it may be impervious (Barbieri), or so small as to be filliform (Barbieri), or it may be unusually short (S. G. Weber, Barbieri), or it may be abnormally large in diameter (Barbieri), or it may be both abnormally short and abnormally thick. Again, one carotid may furnish both anterior cerebrals (Beaumont, Randall), or the anterior cerebrals may be undivided, that is, they may be represented by a single longitudinal vessel formed by the two carotids, just as the basilar is formed by the two vertebrals (Ebstein). Finally, *there may be a direct anastomosis of the carotids without the intervention of any communicating vessel*. Incoronato described a beautiful instance of such a case. The anterior cerebrals form a large quadrate lacuna or sinus, before they separate.

We have, therefore, in the anomalies of the circle of Willis, especially in its anterior portion, abundant evidence of its variability and abundant justification for the explanation by Dr. Mitchell that we have here an aneurism of an anomalous vessel.

Another point requires passing notice. Regarding division of the optic chiasm by the aneurism, doubt might for a moment arise in our minds as we recall the fact that the optic commissure has been, on some rare occasions, found

wanting. In such a case the optic nerve runs to the eye of its own side without any exchange of fibres with its fellow. That such was not the case in the present instance is shown by the condition of the optic nerves and tracts already detailed. They had undergone marked destructive change, especially in the region corresponding to the chiasm. It is exceedingly improbable that much, if any, destructive change would have occurred in the absence of a chiasm. The nerves would in the beginning have been widely separated, and an aneurism growing between them would in all likelihood have merely displaced them. The clinical history, too, of sharply defined hemianopsia, utterly discountenances this view.

Regarding the manner of the division of the chiasm, it is extremely probable that it occurred from below upwards and from before backwards. A study of the anatomical relations of the parts and of the probable position of the anomalous vessel, make this the only tenable view. Besides, it can be readily understood how gradual and long-continued pressure applied to the chiasm in this position would lead to destruction of the internal bands, and, as a consequence, to the eventual separation of the nerves. That the pressure was not exactly in the middle of the commissure is rendered probable both by the clinical history and by the unequal destruction of the two nerves and tracts.

Excessive care has been taken in the examination of the case, and the utmost caution in our inferences. If, as we believe, our pathological explanation be correct, this case stands alone in pathogeny. Moreover, it is an absolute demonstration of the function of the internal bandelettes in accordance with the views of modern pathologists.

APPENDIX.

A Collection of Anomalies of the Circle of Willis.

Barkow, Blutgefäesse der Menschen, 1866. The anterior communicating artery may be double or may make a plexus, or may give off an accessory anterior cerebral.

Barkow, Schlagad. der Säugethiere, 1866, Taf. 43. The anterior cerebral arteries unite like the vertebrals to form one anterior trunk like the basilar.

Spitzka, communicated by letter [not separately published].

a. Anterior communicating artery absent.

b. Left posterior communicating artery absent.

c. Anterior communicating artery double.

Decker, Sitzsberichts der Würzb. Phys. Med. Gesellschaft vii., Stsg. No. 3, S. 33-43. [*Virchow-Hirsch.*] describes an anomalous vessel springing from the cavernous carotid and joining the basilar. Both vertebrals are exceedingly small, but both posterior communicating, very large and form the main supply of the basilar. They are also directly continuous with the posterior cerebral.

Barbieri, Agostino, Monografia della arteria vertebrale, Milano, 1867-68. The following brief abstracts were made from twenty cases collected by Barbieri:

CASE 1.—Impervious anterior communicating artery. Basilar artery nine m.m. in size. Branches of the carotid comprise the posterior communicating, which are rather pronounced.

CASE 2.—Short and large anterior communicating.

CASE 3.—Absence of anterior communicating. Anterior cerebral and posterior communicating on left side a little larger than ordinary. The posterior cerebral of left side smaller than the right and having the appearance of a double origin, in part from the internal carotid and in part from the basilar.

CASE 4.—Double anterior communicating. Left anterior cerebral natural, and right anterior cerebral a third larger than normal. Left posterior cerebral originates equally from internal carotid and basilar. Right posterior cerebral comes in greatest part from internal carotid. Both are very large.

CASE 5.—Anterior communicating short and large. Left anterior cerebral much larger than normal. Right anterior cerebral scarcely a little smaller than normal.

The posterior communicating arteries are merely branches extending backward from the carotid and after

being joined by small branches from the basilar they continue on in the ordinary course of the posterior cerebrals.

CASE 6.—Anterior communicating filiform. Left posterior communicating is of mixed origin arising both from carotid and basilar.

Right posterior communicating arises from carotid and communicates with basilar by means of an excessively slender twig.

CASE 7.—Anterior half of Circle of Willis well developed. Anterior communicating of ordinary length, but a little greater in calibre.

Right posterior cerebral originates from right carotid and communicates by a thread-like posterior communicating with the basilar.

Left posterior communicating, filiform and impervious.

Left posterior cerebral given off by the basilar.

CASE 8.—Anterior communicating normal. Right posterior communicating slender. Left posterior communicating normal.

CASE 9.—Anterior communicating normal. Anterior half of Circle of Willis better developed than the posterior. Right posterior communicating slender, and left posterior communicating a little larger than normal.

Left posterior cerebral originates equally from the carotid and the basilar.

CASE 10.—Circle of Willis as a whole small. Superiority of carotid over vertebral branches. Left posterior communicating quite contracted. Posterior cerebrals are equal in first part of course, but after being joined by the posterior communicating, the right receives a greater blood current than the left.

CASE 11.—Circle of Willis asymmetrical through shortening of its left half. Anterior communicating large and short. An abnormal branch runs from anterior communicating forward and constituting a third anterior cerebral and equal in calibre to the other two [Wilder's "Terminal"?].

Right posterior communicating long, thin and impervious.

Left posterior communicating quite short and a little larger than normal.

CASE 12.—Both posterior communicating filiform.

CASE 13.—Both posterior communicating filiform. Carotid branches more pronounced than the vertebral branches.

CASE 14.—Anterior communicating very short and large. Assymetry of Circle of Willis. Left side complete and much developed. Right side incomplete and poorly developed.

Right anterior cerebral constricted previous to giving off of anterior communicating ; thence normal in size.

Left anterior cerebral one-third larger than normal previous to giving off of anterior communicating and continues to be large afterward.

Right posterior communicating wanting. A minute twig springs from the right posterior cerebral at the part at which the posterior communicating is given off and extends for about two-thirds the usual course of the latter, when it breaks up into a number of minute branches. These are lost in the pia mater and brain tissue with the exception of a few that anastomose with small capillary branches given off by the right carotid.

Left posterior communicating, normal.

CASE 15.—Circle of Willis poorly developed, especially in posterior half.

Anterior communicating quite large and short. Left posterior communicating wanting, the right filiform.

CASE 16.—Anterior communicating slightly shorter and larger than usual.

Left posterior communicating wanting, replaced by minute branching twigs. Right posterior slightly narrowed.

CASE 17.—Anterior communicating of normal calibre, but rather short.

Right posterior communicating wanting, replaced by minute twigs.

Left posterior communicating larger than ordinary.

CASE 18.—Anterior communicating quite large and rather short.

Absence of both posterior communicating, small twigs in their places.

CASE 19.—Absence of posterior communicating, replaced by small twigs.

CASE 20.—Absence of left posterior communicating and filamentous condition of the right. Extremely filamentous anterior communicating.

Anterior cerebrals and posterior cerebrals very small.

Beaumontoir, Le Progrès Médical, 1886, p. 191, describes three anomalies:

1. The basilar furnishes a right posterior cerebral, but does not furnish a left posterior cerebral. This is supplied by the left internal carotid.

2. Left internal carotid furnishes a posterior cerebral, and this anastomoses with the basilar. It also gives off the middle cerebral, but no anterior cerebral. This comes from the right internal carotid. A minute vessel forms an anastomosis between the left carotid and the left anterior cerebral.

3. The right internal carotid gives off a middle cerebral, which, after a course of 18 m.m., divides into two branches which constitute the right and left anterior cerebrals.

Flesch, Verhandlung der Phys. Med. Gesellschaft zu Würzburg, x.—Virchow-Hirsch, Jahresbericht, 1886, ii. 12, describes a case in which the circle of Willis remains incomplete because of the absence of the posterior communicating artery.

Randall, "Unusual Abnormalities of the Arteries at the Base of the Brain," Journal of Anatomy and Phys., London vol. xiii., p. 396, 1886. The vertebral arteries of either side were joined as usual to constitute the basillar, which immediately after its formation divided into two trunks, and again uniting formed a loop. The loop, which was about two lines in length, was situated near the lower border of the pons Varolii. The main artery now ran forward, giving off the inferior cerebellar and transverse arteries of the pons, also the two superior cerebellar arteries, and then terminated in two small branches in the position of the posterior communicating arteries.

Each internal carotid, after giving off the ophthalmic branch, varied in its arrangement on the two sides. The left carotid gave off first the posterior cerebral artery. This was joined by the small communicating branch from the basilar, and then ran on to its normal distribution in the posterior lobe of the cerebrum. The next branch was the middle cerebral, which was quite normal both in size and course.

The terminal portion of the left internal carotid having the direction and appearance of a large anterior cerebral artery, ran forward to the anterior extremity of the locus perforatus anticus, where it divided into two equal branches. One of these, in direction and appearance the terminal of this vessel, continued forwards in the usual course of the left anterior cerebral artery. The other division took a sharp turn to the right, and then, after a short transverse course of about two and one-half lines, another sharp turn forwards, so as to almost form two right angles where it constituted the anterior cerebral artery of the right side.

The right internal carotid artery resembled the left in giving off both posterior and middle cerebrals. These two arteries had the usual size and distribution. The former received the communicating branch from the right basilar. As the fundamental right anterior cerebral was derived from the left internal carotid, the branch of the right carotid which represented that artery was very slender and joined the functional vessel where it passed forwards into the great longitudinal fissure.

Incoronato, Di un anomalia del polygono arterioso cerebrale. Recherche fatte nel laboratorio de Anatomia normale della R. Universita di Roma, nell, anno 1872, p. 95, describes an anomaly³ of the Circle of Willis, in which the carotids unite *directly* with one another instead of doing so by means of the anterior cerebrals and anterior communicating. The posterior communicating are wanting.

³ In this case aneurismal disease at the seat of anostomosis would have inevitably interfered with the chiasm, and in all probability with eye symptoms resembling those in Dr. Mitchell's case.—DERCUM.

Incoronato Anomalia del polygono arteriosa cerebrale, *Atti Acad. Med. de Roma*, 1878, vol. *iu.*, fasc 2, 16-24, describes an anomaly in which there is a double anterior communicating artery, the anterior being much the larger of the two. Again, the right cavernous carotid instead of communicating with the posterior cerebral by means of a posterior communicating artery, anastomoses by means of a large trunk directly with the basilar which it joins at a point corresponding to the normal division of the latter into the two posterior cerebrals.

Weber, S. G., *Abnormal Distribution of Circle of Willis*, *Bost. Med. and Surg. Jour.*, vol., *cvii.*, 543, 1882. The two anterior cerebral arteries came so near each other that the anterior communicating was only about one-tenth the usual diameter. The right posterior cerebral artery was twice the usual size or larger.

The left posterior communicating artery was nearly twice the usual size and virtually was the origin of the posterior cerebral. The left posterior cerebral was about half the usual size until it met the posterior communicating. Thus the region supplied by the right cerebral artery received nearly all its blood through the basilar; that supplied by the left posterior artery received more than three-fourths its blood through the left carotid.

The right posterior cerebral was so small that it could not be distinguished from other small and terminal arteries. Its place was taken by one of the branches from the basilar which was larger than usual, and after passing backwards and upwards, occupied the normal position on the posterior cerebellar between the cerebellum and medulla. The basilar artery was larger than usual, the vertebral uniting at a lower level than normal.

The middle cerebral arteries and the anterior cerebral arteries were normal.

ON GOLD AS A STAINING AGENT FOR NERVE TISSUE.

BY DR. HENRY S. UPSON,

OF CLEVELAND, O.

(*Second Paper.*)

IN staining nerve structures with chloride of gold, as was pointed out in the previous paper on this subject, the differentiation takes place mainly during the process of hardening, so that it is necessary that this should be conducted with extreme care. At the same time, the treatment with the reducing fluid is in a certain sense the pivotal process, as it is at this time that the section takes the actual stain.

With the process given in that paper, very little depends on the way of manipulating the sodium hydrate solution or the reducing fluid; I believe that with ordinary care the stain obtained varies accurately with the differences in the tissue and in the hardening process. The factors of which we must take account in hardening are: First, the length of time which elapses between death and the bringing of the tissues into Müller's fluid. Second, the temperature at which the process is conducted. Third, the length of time in Müller's fluid. Fourth, the length of time in each of the successive reagents during the imbedding and cutting process, and, after this, before staining.

It has long been an object with the profession to find a stain or, at any rate, a chemical test capable of showing changes in the tissues too subtle to result in a change in the form of the elements. If, as seems probable, some or all functional diseases are the expression of molecular or chemical variations from the normal, this is an advance which may reasonably be expected. It is evident that a method delicate enough to accomplish this purpose must also show

such marked changes as occur in the body within a moderate time after death.

From my observations on gold staining thus far, I think that post-mortem changes are shown by an increased liability of the myeline sheaths and neuroglia to take the stain, making such changed tissues harder to differentiate.

If in pathological specimens we can eliminate this source of error by bringing the tissues fresh into Müller's fluid, if they are then hardened at a fixed temperature and under identical conditions with normal tissue, then any greater tendency on the part of the myeline sheaths of the tissue in question to take the stain will be evidence of ante-mortem change.

It is often impossible to obtain pathological specimens until after post-mortem changes have begun. It may then be necessary, before sections are brought into the gold chloride solution, to bring them for a day or two into a four per cent. alcoholic solution of sulphuric acid.

The following method will be found better in most respects than the preceding one:

Method No. 2.—To 30 c. c. of a 1 per cent. tincture of iodine add 1 gramme of proto-chloride of tin, and label this tin solution.

Make a 5 per cent. solution of phosphate of iron, and label this iron solution; the scale salt should be used, as prepared for medicinal purposes.

Tissues should be hardened in Müller's fluid and in alcohol, and the sections cut and preserved, as in the preceding method.

The section to be stained is brought for half an hour or more into a 1 per cent. solution of gold chloride; it is then washed for a moment in water, and brought for half a minute or more into a 10 per cent. solution of sodium hydrate, to which has been added one-half per cent. or less of ammonium vanadate; it is then washed in water and brought into the reducing fluid freshly made as follows:

Tin solution,	gtt. xv.
Distilled water,	c. c. iii.
Iron solution,	gtt. iii.
Sulphurous acid,	c. c. iii.

The section in a few moments takes a somewhat metallic purple color ; it is then washed in water and mounted in Canada balsam as usual.

Ganglion cells and axis cylinders are sharply stained, and the endothelium of the smaller blood-vessels is apt to be stained, showing the vessels in outline ; myeline sheaths, connective-tissue nuclei, and glia tissue are for the most part unstained ; the glia tissue in the posterior horns and around the central canal is most apt to be colored.

This method may be varied almost indefinitely.

Chromic acid may be added to the sodium hydrate solution in place of ammonium vanadate. A mere trace will be sufficient. The stain is then more reddish in color.

Iodide of tin may be bought as such, or made by adding either tincture of iodine or potassium iodide to tin protochloride.

Ferrous sulphate or ferric nitrate may be used instead of the phosphate of iron.

Nitrate of tin gives a remarkably clear picture of the ganglion cells, but is difficult to obtain.

This method requires comparatively little oxide of chromium in the tissues, and gives a clear stain in sections after a much longer stay in alcohol than is possible with the first method.

If the tissues still contain too much of the chrome salt, sections should be brought for a day or two into a 4 per cent. solution of sulphuric acid before staining.

The above methods depend on two principles :

First.—The reduction of metallic salts ; this may happen by the abstraction of the metal or metallic oxide by the tissue, oxygen being set free, or by the abstraction of oxygen by a reducing agent, leaving a lower oxide of the metal combined with the tissues. Thus, in hardening, potassium bichromate ($K_2CrD_4CrO_3$) is in solution ; it is decomposed, chromium trioxide (CrO_3) is taken up by the tissues either in this form or as chromium dioxide (CrO_2^*), which gives to the tissues a brown color ; later, more oxygen is lost, leaving chromium sesquioxide (Cr_2O_3), which gives to the tissues a green color ; potassium chromate

(K_2CrO_4) remains in solution and gives to the hardening fluid a brown color instead of the original yellow.

Second.—The substitution of one metallic salt for another in an insoluble compound. The chromic salt, either the dioxide or the sesquioxide, is replaced by the gold salt, which is then reduced by further manipulation.

Gold replaces silver in a similar way, with this difference: in a section stained by the Golgi or other silver method and brought into gold chloride solution, the purple oxide of gold is deposited at once, following accurately the lines of the silver salt. In this way the stain is rendered sharper and more durable. If now a silver specimen treated in this way, instead of being mounted at once, is brought successively into sodium hydrate solution, and either potassium iodide solution or the reducing fluid given in the previous paper, the unstained parts take a bright red color, thus giving a double gold stain; the original silver stain is in purple, everything else in red. This, with some other observed facts, tends to show that in hardening with Müller's fluid all tissues are impregnated with chrome salts, and that these are replaced throughout by gold or silver salts, as the case may be. The differentiation results from an unequal reduction due largely to a difference in the form in which the chrome salts are deposited in the different tissues.

The purple color resulting from the treatment of gold chloride with a tin salt is called the purple of Cassius. In this case it is probable that the oxide of vanadium and the oxide of chromium respectively enter into the formation of the color; whether there is also present any oxide of tin is very doubtful.

Reviews.

THE LANGUAGE OF MEDICINE. By F. R. Campbell, A.M., M.D. D. Appleton & Co., 1888. Pp. 318.

Although the object of this volume is, as the author states, "to provide the medical student with a suitable means of acquiring the vocabulary of his science," it is, unless the term "medical student" is used in a general sense, more likely to prove of greater advantage to the fully developed practitioner, and will undoubtedly be greatly appreciated by the profession. A work of this description has long been needed, and it is but justice to the author to state that his production in a general manner fulfills all requirements. There are very few, if any, of our medical lecturers, and they comprise the better educated part of the profession, who in the course of their discourses do not pronounce some of the most common medical terms incorrectly. There are very few physicians who can pronounce the names of half the drugs they employ in strict accordance with the principles of orthœpy, and but a limited number of our practitioners could faultlessly write the prescriptions they are in the habit of giving daily if it were necessary to add the terminations to the words they habitually abbreviate. The fault lies often at the door of an incomplete elementary education, but unfortunately, even with the better educated, there is a tendency to pronounce words as they hear them pronounced and to show a profound indifference whether the words they use are pronounced in strict accordance with fixed rules or not, so long as they are understood by the individuals to whom they are addressed. It is at once interesting and instructive, in connection with this subject, to read, on page 66, the medical address delivered in imagination to the ancient class of Dr. Hippocrates by a supposititious professor of a modern medical college. It is just such an address as hundreds of our students are listening to every day. It is not overdrawn or exaggerated, and yet the errors in it are voluminous.

The work treats of the Origin of the Language of Medicine, the Latin Element, the Greek Element, and the Elements Derived from the Modern Languages.

Under these general headings the subjects of orthography, orthoepy, the various parts of speech and the prefixes and postfixes are discussed and explained in a highly satisfactory manner. In addition, there is a list of terms commonly mispronounced, together with their correct pronunciation, and a chapter on prescription writing, which leaves little to be desired. The author is to be congratulated upon having written a book which can be read with both pleasure and profit by the profession.

PHYSICIANS' INTERPRETER IN FOUR LANGUAGES. By
M. VON V. Pp. 206. T. A. Davis, Philadelphia, 1888.

This little volume is undoubtedly intended to aid the hospital and dispensary physician in examining the vast number of patients of foreign birth who are annually treated at our public institutions. It is written in four languages—English, French, German, and Italian—and contains those phrases especially pertaining to diagnosis.

It is unquestionable that a knowledge of languages is of decided benefit in hospital and dispensary work, especially in our large cities where the foreign element prefer to congregate. To such of our physicians whose line of work lies in this direction, this little volume will prove of signal service. It would perhaps have been in better taste if the author had had the courage to write his full name upon the title-page.

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Original Articles.

TWO ADDITIONAL CASES OF HEREDITARY CHOREA.

By WHARTON SINKLER, M.D.

TO Huntington has generally been accorded the credit of first describing hereditary chorea,¹ and writers even speak of the affection as "Huntington's chorea," but in Dunglison's *Practice of Medicine*, second edition, volume second, page 245,² will be found a description of what is no doubt the identical disease. The author gives the following letter :—

Franklin, New York, May 5, 1841.

PROF. R. DUNGLISON :

Dear Sir.—In obedience to your kind request I improve my first leisure since my return home in giving you, in as lucid and satisfactory a manner as possible, an account of a singular affection somewhat common in the southeastern portion of this State, and known among the common people as 'the magrums.' Whence the name originated I do not know, but if it be a corruption of the word 'megrim,' I am at a loss to understand how it ever came to be applied by the vulgar to the disease of which I am speaking, and which has nothing in it analogous to ordinary hemicrania or megrim. It consists essentially in a spasmodic action of all, or nearly all, the voluntary muscles of the system, of involuntary and more or less irregular motions of the extremities, face and trunk. In these involuntary movements the upper part of the air passages occasionally participate, as is witnessed by the 'clucking' sound in the glottis,

¹Med. and Surg. Reporter, Phila., Ap. 15th, 1872.

²Lea & Blanchard, Phila., 1884.

and in a manifest impediment to the powers of speech. The expression of the countenance and general appearance of the patient are very much such as are described as characteristic of chorea.

“The disease is markedly hereditary, and is most common among the lower classes, though cases of it are not unfrequent among those who, by industry and temperance, have raised themselves to a respectable rank in society. These involuntary movements of the face, neck, extremities and body cease entirely during sleep.

“This singular disease rarely, very rarely indeed, makes its appearance before adult life, and attacks after forty-five years of age are also very rare. When once it has appeared, however, it clings to its suffering victim with unrelenting tenacity until death comes to his relief. It very rarely or never ceases while life lasts.

“The first indications of its appearance are spasmodic twitchings of the extremities, generally of the fingers, which gradually extend and involve all the voluntary muscles. This derangement of muscular action is by no means uniform; in some it exists to a greater, in others to a less extent, but in all cases it gradually induces a state of more or less perfect dementia.

“When speaking of the manifestly hereditary nature of the disease, I should perhaps have remarked that I have never known a case of it to occur in a patient, one or both of whose ancestors were not, within the third generation at farthest, the subject of this distressing malady.

“It may not be amiss to state that the last person who came under my observation, and who had the reputation of being an honest man, informed me that, in his own case, this involuntary action of the muscles ceased under the influence of all instrumental music, *except that of the common ‘Jew’s-harp.’* I very much regret that it was not in my power to test the truth of this statement.

“I also regret that it is not in my power to give any information as to the condition of the catamenia in those laboring under it. I hope to be able to institute a course of inquiry upon this subject during the summer or fall. The disease is markedly hereditary.

“I have thus, dear sir, given you a general, though perhaps no very lucid and satisfactory account of this singular malady. I may observe that, although the description of chorea in the books apply very well to this disease, it nevertheless seems to differ in several respects from ordinary chorea. 1st. It rarely occurs before middle age. 2d. It never ceases spontaneously. 3d. When fully developed it wants the paroxysmal character.

“After all, may not this disease be a peculiar modification of chorea? Is not its pathology in the main the same, and would it not probably be found to yield to the treatment most suited to chorea, if to any.

I am, dear sir, respectfully,

Your obedient servant,

C. O. WATERS.

Dr. Clarence King is the only writer I have seen who refers to this communication.³ I cannot help thinking that the cases referred to by Dr. Waters are in all probability the same which Huntington has reported. The latter says that he, his father and grandfather have known families in Long Island for generations in which this form of chorea has lasted. Dr. Waters speaks of the disease being common in the southeastern portion of the State of New York, and this would correspond with the locality of Long Island, and the character of the cases is remarkably like those of Huntington's.

Another paper on hereditary chorea appeared in 1863, and this, strangely enough, seems to have escaped the attention of Dr. Huntington and the writers on the subject who have followed him. These cases are from New York, and may also be from Long Island, but as no name or addresses are given it is impossible to trace any connection between them.

The paper is as follows:—

CHRONIC HEREDITARY CHOREA.⁴

By IRVING W. LYON, M.D.

“The writer has been familiar from childhood with a type of chorea so unlike in its *origin* to anything described in our standard text books, that the publication of a few facts in relation thereto has been thought advisable, not only as a matter of interest to the reader, but more especially for the purpose of eliciting the observations of any who have met with indications of kindred significance. The peculiarity of origin claimed for this type consists in its hereditary trans-

³N. Y. Med. Journal, Ap., 1885.

⁴American Med. Times, Dec. 19th, 1863.

missibility; this claim we will endeavor to support by the following facts and considerations.

"The disease, as we have been accustomed to observe it, has been known in the community by the name of *megrimms*. Of the origin or derivation of this term but little can be ascertained, except the conjecture that it may be a corruption of *megrain*, which word, to say the least, is very inexpressive of any leading character of the malady, which is chorea in toto, consisting of 'irregular action of the voluntary muscles when stimulated by the will,' and marked by an obstinate chronicity. It is confined almost exclusively to certain families, so that such are popularly denominated as "Megrim families," and the children of parents afflicted with this disease are very liable to become the subjects of its manifestation, and in turn transmit it to their offspring. So strong is the conviction of its hereditary influence that the people among whom it occurs believed this to constitute a very legitimate method of propagation, and acting accordingly have repeatedly been known to interdict marriage alliances between their children and those believed to be tainted with the megrim diathesis, under the severe penalties of disinheritance and social ostracism.

"Aged and very intelligent medical gentlemen, who have practiced for the greater part of their professional lives in communities where the so-called "megrimms" prevails, testify that they entertain no doubt of its hereditary communicability. We subjoin the histories of three cases.

CASE I.—Mr. A, residing in the town of —, county of —, New York, has well marked chorea, which is general, so that he is constantly, when awake, making irregular movements with the upper and lower extremities, facial muscles, and more or less with those of the body. This condition has existed for many years, but seems not to interfere materially with his general health. The digestive function being well performed. Mr. A has two brothers and three sisters; the two brothers have themselves never had any choreal symptoms, but one of them has two children in

whom well defined chorea has existed for many years ; of three sisters two have had chorea for most of their lives, being now passed middle age. The progenitors of Mr. A on the male side were perfectly free from chorea, but not so on the maternal side—his mother had well developed choreal manifestations from early life, which continued until her decease ; she had also a brother who died during adult life from the severity of the disease. But to go still further, both the grandfather and the great grandfather of Mr. A, on the maternal side, had the same disorder which we find in the children. Whether the collateral instance of the affliction occurred in the families we are not advised.

CASE II.—Mrs. K, town of —, Conn., and a descendant from a family which has long been known and designated as “megrin,” had chorea the most of her life, being about seventy-five years old at the date of her death. She had a family of two sons and three daughters ; of these one son and two daughters had chorea, with which disease they attained an advanced age. No satisfactory information can be readily obtained in relation to the offspring of the son and one of these daughters so affected ; but the other daughter married and had a son, who is now forty years of age, in whom chorea has exhibited itself from puberty.

CASE III.—Mrs. W, formerly a resident of —, county of —, New York, had chronic chorea, and lived to an advanced age. She stated that her mother was afflicted with the same disease, together with her mother’s father. To these cases many more might be added were it deemed necessary to establish the claim premised.”

Huntington’s observations, which first attracted the attention of the medical world to hereditary chorea, were published in a paper on “Chorea,” which he read before the Meigs and Mason Co.’s Academy of Medicine, Middleport, Ohio, on February 15th, 1872. At the close of the paper he describes a form of chorea, which he says, as far as he knows, is peculiar to the eastern end of Long Island. He, his father

and grandfather, whose medical experience had together covered a period of seventy-eight years, had observed for years certain families in which chorea had retained its hold for generations. The chorea presented features which made it distinct and different from chorea as usually met with, and he also mentions that the common form of chorea is extremely rare in that part of Long Island, neither he, his father nor grandfather ever having met with a case there. The features which he particularly points out in the chorea, as seen in the families he describes, are these :—

1st. That it is undoubtedly hereditary. That if a member of one of these families escape the disease the chain is broken and their descendants are free from it.

2d. That the disease is incurable, and that it is likely to be complicated with insanity, a tendency to suicide often occurring in those afflicted with it.

3d. That the disease never occurs before thirty years and seldom after forty.

He mentions two brothers of about fifty years of age, who, although in an advanced stage of the disease and married men, were strongly addicted to flirting with every young woman they would meet, and seemed to see no impropriety in it.

It is to be regretted that Huntington did not give a fuller account of his patients and their family history. It would have added greatly to the value of his paper had he tabulated his cases and traced them back through as many generations as possible.

It is interesting to note that it is in the same part of Long Island where hereditary chorea is found that tetanus prevails to so great an extent.⁵

Since Huntington's observations were published but nine other writers have reported cases in this country and in Europe. The first of these is Landouzy,⁶ who reported the

⁵Hamilton, "Diseases of the Nervous System," p. 375.

⁶Soc de biologie, 1873, quoted by M. Lannois; *Revue de med.* Aug 10th, 1888, p. 647.

case of a man of thirty-seven years of age. His father and one of his sister's died of the same affection, and one other sister had been treated for some "analogous symptoms."

Ewald⁷ met with two cases of this variety of chorea.

CASE I.—Mrs. Kratz, aged fifty years, in whom the disease began at thirty-five years. The irregular movements affected the head, face, arms and legs. The speech was clear and easily understood. When at rest the movements were not so great, but when she was excited they increased in intensity. They were controlled to some extent when the patient fixed her attention on any object, as for example in taking a glass of water to the mouth.

The patient's mother suffered from the same disease and died at the age of fifty-eight. She has two brothers and one sister; the brothers are healthy, but the sister, who is fifty-two years of age, has chorea. She has one daughter of thirty years who is so far healthy. The patient has three children who are not yet old enough to have the disease. She knows nothing of her grandparents.

CASE II.—Auguste Otto, aged thirty-seven. She was healthy until thirty years of age when the disease made its appearance. Her grandmother, mother and five uncles died with St. Vitus' dance. She has had five brothers and sisters, of these two are dead and one sister, aged fifty, is afflicted with the same disease.

The next writer on the subject is Clarence King, M.D., who reports a case in an inaugural thesis⁸ in 1885. He does not give the locality in which the family lived.

He says: "There has come to my notice a family in which this disease has prevailed for at least four generations, and from which most of the observations of my own are drawn. The first patient known to have had hereditary chorea was the great grandfather of the patients now living.

⁷Zeitschr. f. Klin. Med., 1884, Bd. vii.

⁸New York Med. Journal, 1885, p. 468.

To him were born ten children, four of whom became sufferers from this disease. Concerning three of this number I am unable to obtain any history further than that they each had children, who finally developed hereditary chorea. Of the fourth child the history is complete. He had nine children, and only one of the number had the disease. Of the eight children who escaped, each has had children, and, although they have all reached the age at which the symptoms usually appear, none of them show any indications of a choreic character. But to the unfortunate one of the family who had the disease five children were born, three of whom were attacked and are the patients now suffering from it. These three patients have no children, and, in all probability, upon their death the disease will become exterminated, at least so far as this branch of the original family is concerned. The two other children are married, but their progeny have not reached the age at which hereditary chorea is developed. Since writing the foregoing I have learned that a brother of the three patients I have cited has begun to show evidence of chorea. This gentleman, when a child, had the ordinary form of chorea, from which, however, he made a perfect recovery. He is now about thirty-five years of age."

King reported another case⁹ in 1886. The patient was a male, *aet.* fifty-six. He was supposed to be suffering with insanity, but upon examination was found to have hereditary chorea in an advanced stage. The affection had been slowly developing for more than fifteen years. The patient's father died from chorea at sixty-five years of age, having suffered from it for many years. None of the father's brothers or sisters had chorea, but his father died between fifty-five and sixty years of age from the disease. He had been affected for many years. The patient had one half-brother and ten sisters, one sister *aet.* about sixty has had chorea for many years. The others have escaped, although they have passed the age at which this form of chorea usually begins. The patient is the father of four children, the eldest of whom is twenty-five years.

⁹ *Med. Press of Western New York*, Dec. 1886, Vol. 1, p. 674.

Peretti,¹⁰ in 1885, reports a very interesting case with a complete table covering four generations. A Mrs. N, whose parents and grandparents suffered partly from chorea and psychical disturbances, had choreic movements. She had four children, two of whom, Anton N and Mrs. A, had chorea. Anton N died at fifty-two, the choreic movements having begun when he was forty. During the last seven years of his life he had disturbance of his mind. He had seven children by his first wife, and three by his second—four children by the first marriage and two by the second had chorea. Mrs. A. died at fifty-seven, and for many years before her death she had choreic movements. She had five children, of whom three became choreic after the age of forty. One of these children married, and had three children, who were living at the ages of forty-seven, forty and thirty-five and were free from the disease.

Huber has written a paper¹¹ reviewing the subject of hereditary chorea from the time of Huntington's original paper.

Huber's patient was Jacob Rinderknecht, æt 38, silk dyer. The patient was first affected eight years ago; "he awaited his fate with quietness, as he 'knew it was coming.'"

"Status præsens, Oct 8th, 1886. In bed. Head never at rest. Movements regular. The muscles of expression constantly change. Patient can with difficulty hold his mouth open. Cannot whistle. Speech affected. The patient, when ordered, can make exact movements—as to put a finger on the end of his nose. All the muscles are more or less affected. Sensibility intact. No pain. Urine normal. Can fix eyes and follow the direction of a finger.

Family history—Father died insane at fifty-eight years old, having had the same disease as the patient. There were also affected one brother and one sister of the father, also the patient's grandfather and great grandfather. A sister of the patient's had chorea and was seen Nov. 19th, 1886. Elizabeth Wegman, neé Rinderknecht, æt. forty-two,

¹⁰ Berliner Klinische Wochenschrift, No. 50 and 51, 1885.

¹¹ Virch. Arch. c. viii S. 267, 1887; also Amer. Journal Med. Sc., Oct. 1887.

has a stupid look. Head in constant motion, eyes half shut. In trying to speak, she gives rise to indistinguishable expiratory sounds. Tongue hypertrophied—cannot protrude it. Patient hears well, pupils equal and react to light, gait peculiar, movements cease entirely during sleep. In 1873 she married, and in the same year gave birth to a child. From this time her husband noticed that she was slower in her work and indifferent. In January, 1877, she gave birth to another child, which she did not nurse. She was indifferent, unruly and forgetful. In 1880, when thirty-six years old, her husband first noticed abnormal movements of the elbow, then the arm and leg became affected. In 1881 a change in speech was noticed. In the case of the father of these patients an autopsy was made, and pachymeningitis and internal meningitis were found."

The "collective investigation committee of the British Medical Association" reports in the *British Med. Journal*, Feb. 26th, 1887, the following cases observed by Mr. West, of Stoke-on-Trent.

"Charles P., Jr., son of James P., choreic. Stephen, brother of Charles, died at forty; had chorea seven or eight years; was married; his children were unaffected. Charles P., brother of James and uncle of Stephen and Charles, Jr., died of the same disease, aged fifty-one; was affected about ten years. Charles P. had three daughters, one married. Two are quite healthy but very emotional. The youngest is becoming heavy in manner and cross-tempered, just as most of the others who have been affected have begun. She is thirty-three but not choreic at present. The children of the married daughter are healthy. William, a brother of Charles and James, had a daughter who died of this disease at the age of fifty-six, in America. The father of Charles and James lived to a great age and was quite free. His father lived to over ninety and was free from it. The mother of Charles and James was a Wedgewood, of Etruria, and had it some years before death. It began when she was almost sixty, and was caused by a fright. Other mem-

bers of her family were given to 'shaking,' and here the first indication of the hereditary taint seems to have begun."

Mr. West expressed the opinion that the disease was "organic and not the ordinary functional chorea."

In 1884 Mr. West¹² reported the cases of two brothers who had chorea; one died at fifty and the other was still living at forty-five. There was no history of any hereditary transmission in these cases.

McLeod¹³ also reported three cases of chorea in adults, two of whom were sisters. There is no reason for considering these cases of hereditary chorea, as there was no family history of the disease, and each of the cases a post-mortem examination revealed a lesion of the motor area of the cerebral hemispheres.

Zacher (*Neurolog. Centralbl.*, 1888, No. 2, and *Am. Jour. Med. Sc.*, April, 1888, reports a case of hereditary chorea.

Patient forty-five years old, well until four years ago, when disturbance of motion began to develop. For two years he had been unable to walk, and had been very excitable and irritable, with a tendency to destroy everything, and was finally committed to an asylum. He exhibits peculiar, irregular, purposeless movements, apparently in all the voluntary muscles. The head is turned from side to side; the face undergoes all sorts of grimaces. Respiration occasionally has a sobbing, rasping sound. The arms are in constant choreiform movement, and the same motion in the legs renders the gait irregular and laborious. The patient is able to control the muscular unrest for a moment and to perform an intended movement with considerable promptness and certainty. He can write his own name well the first time, but a second immediate trial is not successful. The tongue twists about within the mouth and can only be protruded with difficulty. Speech is rather monotonous and with frequent pauses. All movements cease during the sleep. The muscles are well developed and their strength

¹² *British Med. Journal*, Jan. 5th, 1884.

¹³ *Journal Mental Science*, July, 1881, p. 194.

preserved; patella reflex not increased, and sensibility undisturbed. Intelligence seems slight, though it is difficult to determine how far this is due to his imperfect education. The maternal grandfather and two of his brothers had the "shaking disease," while a sister was insane and died in an asylum. The grandfather had two sons and one daughter. The latter and one son had the same affection, the other son died away from home and nothing is known of his condition. The daughter had seven children, of whom three are now living and were attacked by the disease after the age of forty; one being the patient under consideration. The patient himself has four children, all young, of whom one is said to have occasional twitchings of the face.

Various descendants of the brothers of the grandfather are also said to have the same affection, but the author did not succeed in learning anything more definite regarding them. The writer does not refer to Huntington.

Hoffmann (*Virchow's Archiv*, cxi., 3, 513) reports in full four very interesting cases. Three of these included two brothers and a sister, the fourth being a female cousin. All were attacked about the age of thirty to forty years, except one who developed the disease while still at school, having had epilepsy since two to three years of age. The family history is briefly as follows: Two men of the oldest generation were probably affected by the disease. Next the Grandfather, Waldi, was choreic for several years before his death, at fifty-one to fifty-two. It is not certain whether he was mentally affected. Three of his nine children were choreic. The first was attacked at the age of forty, became weak-minded. One of her two children became choreic at ten, developed imbecility later, and died at nineteen. Another of Waldi's children showed signs of chorea at thirty, and later became insane. She had nine children, of whom three were the first three patients mentioned, and two others were attacked by the same disease at the ages of thirty and forty respectively. A third choreic child of Waldi, a son, exhibited the first symptoms at thirty-eight. There was no mental disturbance (?). His daughter is the fourth of the choreic patients whose case is detailed. The disease

therefore extended through four generations and attacked thirteen persons. None of the children of the fifth generation exhibit symptoms, but none are over twenty-four years old. Two of the cases prove that the statement of Huntington is incorrect, that the disease "never begins in youth."

Hoffman then reports another case which developed chorea at forty and also epilepsy at fifty, and whose mother and three sisters had been epileptics. He believes that it is undoubtedly an instance of chronic chorea, and that the absence of psychic disturbance proves nothing to the contrary, since a case of Ewald's exhibited the same peculiarity. As regards the "heredity," it would seem that epilepsy had taken the place of chorea in the predecessors. This epilepsy differed from the usual hereditary form in that it did not develop in youth, but not until twenty-six to fifty years of age. It is interesting to note that in this case chorea followed epilepsy, while in one of the others, as already stated, the order was reversed. The author then discusses the association of chorea with other nervous diseases. Since this case proves an exception to the rule of inheritance, he confers the title "chorea chronica progressiva." He devotes some attention to the possible anatomical seat of the cause of the disease, but sheds no light on the subject.

The most recent case is one reported by Dr. M. Lannois,¹⁴ who has had the kindness to send me his paper, which is by far the most complete and thorough *résumé* of the subject I have seen. Lannois has had the opportunity of studying the cases in six members of the Vey family who were choreic, and he has traced the affection back to the great grandfather.

Jean Sebastian Vey, the great grandfather, had chorea. He had twelve children; of these the history of eight only could be obtained. Five of them had chorea. Joseph Vey, a son of Jean Sebastian, died at fifty-seven years choreic. He had four children, two of whom were choreic. Francois Vey, son of Jean Sebastian, had two children, one of whom

¹⁴ Revue de Med., Aug. 10, 1888.

was choreic. Catharine, daughter of Jean Sebastian, had five children, four of whom were choreic. The symptoms in the six cases seen by Lannois were studied with great care and correspond with those described by all previous observers.

I have had the opportunity of adding two choreic families to those already reported. The first patient came under my care in the nervous wards of the Philadelphia Hospital in March, 1888. The following is his history, for which I am indebted to Dr. Clara T. Dercum, the Resident Physician :

David M., age 43 years ; white ; born in New York State ; residence, Scranton, Pa. ; occupation, clerk ; weight, 120 pounds ; height, 5 feet 3 inches.

The disease with which the patient is afflicted can be traced back to his grandmother's family on his father's side. His grandmother had six children, three sons and three daughters ; of these, two sons and one daughter were afflicted. The patient has no brothers and but one sister, who was afflicted in the same manner as himself. She developed the trouble at thirty-five years of age, and finally died of phthisis. In his uncle's the affection began in his legs, his aunt and sisters developed the movements of the head first. In all the cases it took several years for the trunk and all the extremities to become involved. Recently another one of his aunts, who is confined in an asylum in New York State, developed the disease. One of his cousins who is dead had the same affection, although his father, James M., was one of his grandmother's children who escaped. The affection has always been spoken of by the family as St. Vitus' dance. The disease never develops before the thirty-fifth year of age. The father of the patient was accidentally killed at thirty years of age, dying before the age at which the affection appears. His mother died from the effects of a fall. The different members of the family thus afflicted die from some intercurrent affection.

Previous History.—Has had the usual diseases of childhood. Drinks beer moderately. Has had gonorrhœa.

Seven years ago, at thirty-five years of age, patient developed irregular, involuntary movements of the legs. His gait became hesitating and staggering; it gave him no inconvenience, but it annoyed him, because people imagined him drunk. He first noticed it himself in coming down hill, which was only accomplished with great difficulty. Five years ago, at thirty-seven years of age, the same involuntary movements developed in the arms. Two years ago, at forty years of age, the head and trunk became involved.

Status Præsens.—The patient has choreic movements of the whole body, every muscle being involved in these in-



voluntary movements. The head is being constantly turned from side to side, and the arms, especially the left, are continually in motion; the finger movements are somewhat like those seen in athetosis, as shown in cut. The sterno-cleido-mastoid muscles are considerably hypertrophied, although the other muscles of the body, which are also in constant motion, are not enlarged. All the movements are very much increased if patient is watched, or if he attempt to speak or walk. His speech is difficult on

account of incoordination of muscles. His gait is characteristic, he flexes his legs but slightly, plants one foot firmly on the ground, and rests it a moment before lifting up the other foot. There is no motion during sleep, patient lying perfectly quiet. Tongue is protruded easily, but is almost instantly withdrawn with a quick jerky movement. When sitting the patient grasps the side of the chair to steady and rest himself. He has no pain whatever, and is unconscious of the movements himself unless his attention is attracted by others gazing at him. On making a voluntary effort he is able to control the movements to some extent, but cannot stop them entirely. For example, he can steady his hand enough to write his name in a jerky manner.

A handwritten signature in cursive script, reading "David Fowler". The letters are somewhat shaky and irregular, reflecting the patient's condition.

He is able to hold a book so as to read it, and can read aloud in a peculiar spasmodic way, but the book is in constant motion from the movements of his hands, and the head is also shaking, so it is remarkable that he is able to read. He is unable to whistle. He feeds himself with great difficulty. He cannot cut up his food, and in attempting to carry his food to his mouth scatters it about.

Knee-jerk grossly exaggerated. No ankle clonus. Sensation very acute, almost hyperæsthetic. Eyes have been examined by Dr. de Schweinitz, who has pronounced them, as well as the ocular muscles, normal in every respect. Examination of the urine revealed it to be normal. All organs have been examined, and as far as can be ascertained are normal. Bowels regular, appetite good, altogether his general health is excellent. His memory is fairly good, and he seems to be intelligent. There are no delusions or evidence of insanity. Table I. gives the family history of James M., as far as can be traced. It was through this patient that I became aware of the other family of hereditary chorea, which I will report later. He told me

that at the onset of his troubles he thought that he could keep off the disease by travelling about. He got a peddler's pack and went about the country selling his goods. In his travels he saw members of a family in Wyoming Co., Pennsylvania, who he thought suffered from the same disease as himself. I wrote to the address which he gave me, and through the kindness of a member of the family who has sent me a most intelligent communication, and Dr. C. R. Newton, of Nicholson, Penna., I have been able to get a very good account of the disease.

The first member of the family of whom anything definite is known was Mrs. D., who was choreic. She had a large family; two of her sons and one daughter were "afflicted." One son never married. Another son had three children; neither of these nor their descendants had the disease. The daughter had two daughters, Elizabeth and Polly; both were "afflicted." Elizabeth married a B., and had six sons

TABLE I.—M. Family.

The names of those who were choreic are in *italics*.

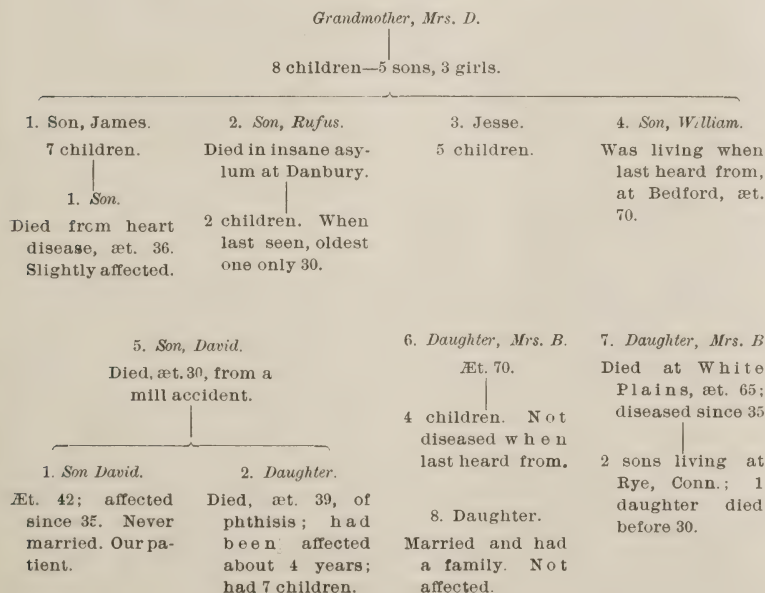


TABLE II.—S. Family. The names of those affected are in *italics*.

Mrs. D. A large family.

1. Son.		2. Son.		3. Daughter.	
Never married.		3 children. They nor their descendants did not have the disease.		1. Daughter, Elizabeth 9 children.	
				2. Daughter, Polly. 9 children.	
				3. Daughter.	
				1. Burton. Died at. 2.	
				2. Prudence. 3 children. Afflicted at 30, died at. 65.	
				3. Dana. Died at. 61. Par- lost his mind. Was not nervous.	
				1. Son living. 2. Son. 3. Son. 4. Son. 5. Son. 6. Son. 7. Daughter. 8. Daughter. 9. Daughter.	
				1. Harnon. Afflicted at 30; very bad.	
				5. Nancy. 3 children. When afflicted, 50; died at. 56.	
				6. Elizabeth Æt. 67. 10 chil- dren. Slightly afflicted.	
				1. Loren. Afflicted at 40; at. 45.	
				Several children show symptoms.	
				8. Perry. Afflicted at 45; died at 61. 2 children. The one who has just died.	
				9. Sarah. Died at 2.	
				4. Lucy. 4 children. Afflic- ed at 40; is now nearly 70.	
				1. Melo (son). Afflicted at 30; now 45.	
				7. Pauline. Æt. 64. 3 children. Slightly afflicted.	

and three daughters; one son has been "afflicted," two of the daughters show symptoms. Polly married a S., and had nine children:

1. Burton S., died at the age of two.

2. Prudence S., had three children; she had chorea at thirty, and died at sixty-three. One of her children, H., was "afflicted" at the age of thirty, and has the disease severely.

3. Dana S., died at sixty-one, partially lost his mind, but was not nervous.

4. Lucy S., four children; had chorea at forty, is now seventy. She has one son, M., "afflicted" at thirty, and is now nearly forty-five.

5. Nancy S., had three children; she was afflicted at fifty and died at sixty-six. One of her children, L., had chorea at forty and is now forty-five.

6. Elizabeth S., had ten children, is sixty-seven years of age, and is slightly choreic. Several of her children show symptoms.

7. Paulina S., had three children; aged sixty-four, and is slightly affected.

8. Perry S., had two children; he became choreic at forty-five and died at sixty-one.

9. Sarah S., died at the age of two years.

Dr. Newton, who attended Perry S. in his last illness, wrote me the following letter:

NICHOLSON, PENN., Sept. 14th, 1888.

Dr. SINKLER:

Dear Sir—Yours in reference to the late Perry S. at hand. I saw him two or three days before he died and one or two times previously. This man had chorea for years. Never knew of any cause for the attack, such as excitement, shock, or fright. It seemed in his case to be hereditary, as several members of his family had chorea. He was considered always a clever, energetic man, and apparently inherited no other disease than the chorea. His movements were of the ordinary choreic form, and gradually became worse until he died from exhaustion—failure of heart. The movements ceased during sleep. The intellect was a little impaired for the last few months. There seemed to be a slight paralysis of

the muscles of mastication and deglutition and phonation, suggesting possibly the seat of the disease. He complained much for months of "pain and pressure in the occipital region."

Yours very truly,

C. R. NEWTON.

In reporting these cases I have no new observations to make. I can only confirm and strengthen what has been said by all who have written before me on the subject. It is most remarkable, however, how little variation there is in the family history and symptoms of the different cases reported. All writers have agreed upon the three main points as set forth by Waters and Huntington:

1. The hereditary nature of the disease.
2. That it occurs between the ages of thirty and fifty.
3. That it is incurable, and that there is often mental disease in connection with it.

Huntington and Clarence King assert that if one of a choreic family escape the disease, his descendants also escape it. Although this is usually the case, it is not an unvarying law, as seen in the instance of one of the M. family which, I have reported. James M. did not have the disease, but one son became choreic. In one of Mr. West's cases it is stated that "William P. had one daughter who died of the disease at the age of fifty-six." It is not asserted that William did not have the disease; but as the fact is mentioned in connection with the other brothers, whether they had the disease or not, it is to be presumed that William did not have it.¹⁵

Another exception to the rule is as to the age at which the affection begins. One of Hoffman's cases¹⁶ developed the disease at ten years, became imbecile, and died at eighteen; and another became choreic "while going to school." One of Lannois' cases had the first symptoms at twenty-one years, and one of Lyon's began "about puberty."

¹⁵ In the A. family of Lyons there were two sons and three daughters. One son and two daughters had chorea. One son escaped, but his son had it. Walters says the disease may pass over a generation.

¹⁶ *Op. cit.*

¹⁷ In one of Hubers's cases, Magdalena R., it is not stated that she was choreic, although her son Heinrich had the disease; but this may have been an oversight.

Still these exceptions do not influence the generally observed fact that the disease, as a rule, does not occur before thirty or after fifty years of age.

Sex exerts but little influence on the disease. In the two families which I have reported there are eleven males and fifteen females in a total of twenty-six cases. Lannois gives nine males and ten females out of nineteen cases. One fact of importance is that the disease does not materially shorten life, although eventually most of the patients die as the result of the affection. Still it is notable the advanced age that many of them attain. Among my own cases are the following ages: Prudence S. died *æt.* sixty-five, Dana S. died *æt.* sixty-one, Lucy S. living nearly seventy, Nancy died *æt.* fifty-six, Elizabeth living *æt.* sixty-seven, Perry S. died *æt.* sixty-one, M. died of heart disease *æt.* thirty-six, William M. living *æt.* seventy, Mrs. B. living *æt.* seventy, Mrs. B—t died *æt.* sixty-five.

Another point which strikes one in reviewing the cases reported is the unusual number of large families met with among them. Among my own cases are one family of eight children, two of nine, and one of ten. In the Vey family of Lannois, the great-grandfather had twelve children, and Francois, his eighth child, had thirteen.

The character of the movements are markedly like Sydenham's chorea, and I cannot accept the point of difference made by Lannois and others that in hereditary chorea the movements are controlled by effort of will or when a voluntary movement is attempted, for in a certain proportion of cases of ordinary chorea, as seen in children, this is also observed. This has been noted by Mitchell, Gowers, and Hamilton. The movements in the chorea of the aged are quite different from those in hereditary chorea. The irregular motions are never so general or so violent.

The knee-jerk in hereditary chorea seems to be exaggerated. In the case of David M. it is greatly increased, and Lannois found it exaggerated in his six patients. In most of the cases of Sydenham's chorea I have examined, the knee-jerk is lessened or absent, although it is sometimes increased.

We must not fail to note the distinction between hereditary transmission in chorea and the hereditary tendency to chorea. The first is rare, while the latter is common. I have under my care now a girl of six years, who is one of six children, five of whom have had chorea.

There have been but few observations made as to the condition of the heart in hereditary chorea. In the case of David M. there was no evidence of valvular disease, but one of his cousins who had chorea is said to have died of heart disease.

There have been no autopsies made in cases of hereditary chorea which throw any light on the pathological anatomy of the disease. Huber states that the father of his patients died choreic, and at the post-mortem there was found a pachymeningitis and meningitis.¹⁸

The affection of the intellect and the persistent and gradually increasing movements point to some structural change in the brain, and no doubt in a short time opportunities will be given for determining the lesions in this most obscure and interesting disease.

We cannot but arrive at the conclusion that, although there are many points in common between this chorea and the ordinary form, still there are differences enough to make them distinct and separate affections.¹⁹

It is more likely that future investigations will show that chorea in the aged is closely allied in its pathological anatomy to the hereditary form of the disease.

I have said nothing as to treatment, because the disease so far has resisted all medication. In the case of David M., hyoscine was administered in large doses, and for a time diminished the severity of the movements. Large doses of arsenic also seemed to lessen them, but there was no material benefit done.

¹⁸ Dr. William Osler, of the University of Pennsylvania, has recently made a complete autopsy in a well-marked case of hereditary chorea and will shortly publish an account of it.

¹⁹ King's case is of interest in this connection, where the patient had chorea in childhood, made a complete recovery, and late in life had the hereditary form.

One thing we should learn, however, from the study of the cases reported, is that something may be done to prevent the onset of the disease in those predisposed to it by inheritance. When the age at which the disease is likely to come on approaches, the patient should be guarded against all excitement and mental strain; in short, he should be placed under such hygienic conditions as we would think necessary for a patient in danger of mental disease.

In conclusion, I must acknowledge valuable aid in the preparation of this paper to my friend, Dr. H. W. Cattell.

A CASE OF LEPTO-MENINGITIS CEREBRI PRESENTING TYPICAL SYMPTOMS OF DISSEMINATED SCLEROSIS.¹

BY LANDON CARTER GRAY, M.D.,

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THE history of this case is as follows: Male, aged —, who gave a history of having had some ten years before specific symptoms consisting of chancre and papula eruption following. Was under my care and treatment at the New York Polyclinic and afterwards in the Hospital for Nervous and Mental Diseases, some three years. During all this time he presented all the symptoms of a case of disseminated sclerosis, and as such he was observed with interest by my former assistants, Dr. B. Sachs and Dr. M. Allen Starr, and I have also repeatedly lectured upon him as an illustration of this disease. To summarize the history, which is very full and minute, as it extends through several years of my case books, it will suffice to say: The tremor was of the voluntary type, this characteristic persisting to the day of death. In sitting still there was none of it. Upon any movement of the muscles, or upon any excitement, it would be observed. This tremor affected both upper and both lower extremities. The tongue also was tremulous. Both the tremor of the tongue and of the extremities was of the jerky kind which is generally seen in well-marked cases of disseminated sclerosis. The muscles of the face were also extremely tremulous when the patient spoke. There was well-marked nystagmus. The speech was of the scanning, jerky variety, which, in the first year of my observation of the patient, did not interfere with articulation further than by rendering it tremulous. The patellar tendon reflexes

¹ Read before the American Neurological Association, Sept. 20, 1888.

were always somewhat exaggerated. When the patient first came to my clinic, these were all the symptoms. His memory was good, as was also his intelligence. The statement made by himself and his wife was that the disease had been gradually coming upon him for a period which they indefinitely expressed as two or three years. There was no history whatsoever of any mental trouble or, indeed, of any alteration in the patient's mental characteristics. There was no history of cephalalgia or trauma. A careful examination of the different cranial nerves, as well as the sensations of tact, pain, temperature, and muscularity in the trunk, head, and limbs, showed nothing abnormal; there was no neuro-retinitis at any time in the course of the case. The man was taken into the wards of the Hospital for Nervous and Mental Diseases, and kept under constant observation for nearly three years. During this time he had a number of apoplectoid attacks, characterized by a temperature up to 102, 103, once 104 even, lasting for twenty-four or thirty-six hours; violent emesis, usually at the beginning of the attack; great dilatation of the capillaries of the skin of the face; a curious chattering of the lower jaw against the upper, that would come on in paroxysms throughout the attack; occasionally a violent generalized convulsion with loss of consciousness, that was sometimes repeated in the same attack. During these attacks the patient would be so weak that he could not rise from the bed, and this condition of motor weakness would last for several days, passing off gradually. Upon no one of these occasions was there any paralysis induced, motor or sensory. During these attacks the patient's face bore an anxious, distressed look, and, over and above the increased tremor of the tongue and face, there was evidently a motor inability of the patient to articulate. For this reason it was impossible to judge as to the amount of mental confusion, the more especially as the patient maintained, after the attack had passed off, that he was perfectly aware of all that had taken place during it. The convulsive phenomena, as well as the chattering of the lower jaw, were seemingly relieved quickly by the administration of bromides and hypodermics of morphia. In the

course of a year the patient's mind very gradually became more and more impaired by passage into a simple dementia, without hallucinations, illusions, or delusions of any kind. A general motor weakness gradually made itself apparent, but never progressed beyond a slight degree until two or three weeks of death; thus, when being brought across in a car on the Brooklyn Bridge, the slight jar usually felt in the train at the moment of stopping was sufficient to cause the man to fall full length upon the floor. At the same time, within six months of his death, he was strong enough motorially to wander out of the hospital, travel several miles around the city, and so effectually lose himself that it took us several days to find him again. Toward the last he grew very much demented, and so obnoxious did he therefore become in his habits that it required almost the sole attention of one nurse. This extreme condition of dementia existed some four months before his death, and yet his motor weakness had not progressed beyond the degree that I have stated, and did not progress until within three weeks of his death, when he remained most of the time in bed, and on attempting to walk was likely to stumble against any object in the way and fall, although even at this time he moved his limbs so freely and persistently that it was almost impossible to keep any clothing upon him. It is probable that the apparent motor weakness of these latter days was due in greater degree to the dementia than to any great injury to the motor fibres. During the period of dementia it was, of course, impossible to make any accurate examination of the different senses, although we could, of course, perceive in a general way that his sight and hearing were good. Death came about by gradual *æsthenia*, and was quiet and painless.

That this case was one of disseminated sclerosis, no one can doubt. The characteristic voluntary tremor, the peculiar scanning speech, and the nystagmus were three symptoms persisting for several years that almost settled the diagnosis of themselves. The differential diagnosis was carefully considered from general paresis and intercranial specific trouble. General paresis was excluded because of the

lack of any characteristic mental symptoms or pupillary irregularity, as well as because the speech and the tremor were not those of this disease ; nor at any time in the course of the disease were there any symptoms that led us to doubt these conclusions. Intracranial specific trouble was excluded because of the lack of any cephalalgia or insomnia, or paralysis or affection of any of the cranial nerves. The typical tremor of the purest voluntary type, the scanning speech, the nystagmus, the age of the patient, the upright gait, and the remarkable persistence of these symptoms until the very last, were certainly facts upon which alone a diagnosis of disseminated sclerosis could be made, unless we are to discard entirely our diagnostic criteria of this disease, as they have been made classic by the descriptions of every writer upon the subject. Paralysis agitans, it is hardly necessary to say, was excluded for the reasons that have been given above, as well as because of the lack of the characteristic attitude and the characteristically deliberate speech.

The autopsy was made twelve hours after death. The blood was very fluid. Brain substance was found to be soft and oedematous. There was a great excess of fluid in the ventricles. The dura mater was normal. There was a severe lepto-meningitis over all the vertex and the cerebrum, extending down on either side to the tempora-sphenoidal lobes, so as to implicate irregularly the second temporal convolutions ; at the base of the brain the pia mater seemed normal over the bases of the frontal lobes and backward over the bases of the cerebrum and cerebellum.

Careful search was made for patches of sclerosis, but none were found ; nor was there at any point the hyperæmia upon contact with the oxygen of the air which has been so often observed in sclerotic patches. Sections were also carefully made of the cerebrum, each section varying in thickness from a half inch to an inch, so as to permit of subsequent microscopical examination ; and especial care was observed in examining around the walls of the cerebral ventricles, but at no point was a sclerotic patch found. The pons,

the medulla oblongata, and the spinal cord were stripped of the dura mater, and their external surfaces also examined, but no evidences of sclerosis were found in any of them. Sections throughout the cerebellum were equally negative.

The only case in any way similar to this which I can find in the medical literature, is one detailed by Koenig before the Berliner Gesellschaft für Psychiatrie und Nervenkrankheiten, Sitzung von Jan. 10, 1887, relating to a man thirty-three years old, who fell in his twelfth year upon the left side of his head, was rendered unconscious, and only became able to walk and speak after several months. Later on there appeared dementia and the following phenomena: dragging of the right leg, slight right facial paresis, distinct evidences of motor aphasia, diminution of the perception of pain on the right side, marked tremor of the extremities upon voluntary motion; patella tendon reflex present on both sides; finally complete dementia and death from pneumonia. At the autopsy there was found, together with old hæmatoma of the dura, chronic lepto-meningitis, hydrocephalus, and atrophy of the convolutions from sclerosis that was also recognizable microscopically. This case and mine are certainly very strong evidence in favor of the view that the tremor of disseminated sclerosis is due to lesion of the cortex or of the underlying white strands. Unger in his recent monograph upon multiple sclerosis in children, and Hyman in his late brochure upon paralysis agitans, call attention to numerous facts supporting this theory. The former reminds us that in the diffuse form of cerebral sclerosis in children, Erb and Steffen and other authors have observed tremor either of the voluntary or permanent type. Larcher reported a case of diffuse sclerosis principally affecting the pons, in which there was no tremor. Westphal treated a case of paralysis agitans in which all four extremities were tremulous. In the course of the disease the patient became hemiplegic, and thenceforward the paralytic remained motionless, whilst the tremor continued in the other limbs. As Hyman observes, this must mean that the lesion inducing hemiplegia also interrupted conduction from the cortex. This was confirmed by finding at the autopsy

a hæmorrhage in the internal capsules. Parkinson also mentions a case in which certain limbs becoming paralyzed ceased to be tremulous, but in which the tremor was renewed as the paralysis disappeared. Grashey describes a patient in whom a general tremor ceased upon the supervention of a light hemi-paresis of the right side. In a late discussion in the New York Neurological Society, Dr. Starr mentioned that the rate of vibration in paralysis agitans was eight per second, as had been demonstrated by Gowers, and that cortical irritation of a moderate degree in monkeys produced muscular tremor also characterized by eight vibrations per second.

DISCUSSION.

Dr. WEBBER referred to a case of disseminated sclerosis in which the tremor was entirely wanting, the case having been diagnosed as one of locomotor ataxia. Tremor was supposed to be produced by sclerosis of the medulla or pons, and the explanation was that the parts producing tremor were not affected. The case of Dr. Gray's was interesting as presenting tremor with a lesion of the cortex.

Dr. BANNISTER considered tremor due to interference with conduction, not to the cortical affection *per se*.

Dr. MILLS thought that those cases should make us more careful in the analysis of symptomatology. Even some symptoms regarded as pathognomonic were probably not due so much to the nature of the lesion as to its location in the cerebro-spinal axis. Tremor shows a want of co-ordination. Disseminated sclerosis might exist in the sensory tracts without tremor. Lepto-meningitis was an irritative lesion which interfered probably with the initiation of impulses.

Dr. PUTNAM suggested that the tremor in Dr. Gray's case was similar to that found in old cases of cerebro-spinal meningitis. This was a species of intention tremor.

Dr. GRAY replied that he had in his mind a picture of the tremor of meningitis, and that it did not correspond to that in his case. He had seen also a continuous tremor in disseminated sclerosis and an intention tremor early in par-

alysis agitans. This man had a voluntary tremor throughout and scanning speech. The question of diagnosis lay between intracranial syphilis, disseminated sclerosis, and general paralysis of the insane. In many cases of general paresis, however, there was no implication of the pia. In his opinion, the lesion of general paresis was an interstitial encephalitis, not a meningo-encephalitis at all. One year ago the speaker had had a case of lepto-meningitis of the whole tempero-sphenoidal lobe. The patient was perfectly sane, loss of memory being the only symptom. In view of these two cases, the speaker inquired what then could be considered the symptoms of lepto-meningitis. The apoplectoid attacks present in the case, the subject of the paper, had been laid down by Charcot as especially characteristic of disseminated sclerosis.

A CONTRIBUTION TO THE STUDY OF MUSCULAR TREMOR.¹

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PATHOLOGICAL motor disturbances present a wide and interesting field for study, too wide and varied for review in a short paper. Hence, grosser movements, such as the choreiform, athetoid and epileptiform will not be considered here. I have a few words to say upon the finer pathological motions known as muscular tremblings or tremors, such as are found in hysteria, neurasthenia, multiple sclerosis, paralysis agitans, morbus Basedowii, senility, and in poisoning by mercury, lead, alcohol, tobacco, etc., many of which resemble the physiological tremors produced by the action of cold upon the body, and of the depressing emotion fear.

The origin of rhythmical motions of all kinds has been a matter for much speculation. For the coarser oscillations of the different forms of eclampsia and chorea the theory ascribing their usual genesis to explosions of force in the cortical cells of the motor areas of the brain is generally accepted. But the origin of the finer tremors is more mysterious, and the formation of a hypothesis concerning them more difficult. Have they their origin in the nervous system or in the muscular tissue itself? If in the nervous system is it peripheral or central?

In physiology we have learned that curara paralyzes the motor nerve endings, and that the sartorius of a curarized frog when dipped in a saline solution will contract rhythmically for several days (Biedemann).²

¹ Read before the American Neurological Association, at Washington, D. C., Sept. 20, 1888.

² Landois and Sterling. Text Book of Human Physiology, 2d ed., p. 510.

The so-called fibrillar contractions³ occur in the muscles of the tongue after section of the hypoglossal nerve (Schiff), and in the muscles of the face after section of the facial nerve. According to Bleuler and Lehmann these may take place for six months in the tongues of rabbits after section of the hypoglossal. Some drugs cause such fibrillar contractions, as aconitin, guanidin, pilocarpin and physostigmin, according to Brunton by irritating the motor nerve-endings, for the contractions are gradually abolished by curara. Such phenomena may be termed idio-muscular. Yet the researches of Gerlach tend to obscure the doctrine of specific muscular irritability by showing that a nerve fibre on penetrating the sarcolemma divides into inter-fibrillar threads, which come into direct relation with the sarcous substance.

Now as regards the physiological action of muscle through the mediation of the nervous system, we learn that a continued voluntary contraction in man consists of a series of single contractions rapidly following each other, frequent intermittent vibrations which reach their maximum when a person shivers (E. Weber).

Tetanus is a condition of continuous vibratory contraction, an accumulation of contractions which follow each other too rapidly for relaxation to take place.

Horsley and Schäfer⁴ have demonstrated that such contractions, whether natural or not, are caused by impulses from the central nervous system along the motor nerves, discharged rhythmically at the rate of ten per second. This innervation rhythm may be generated in the motor cells of the spinal cord, in the medulla, pons or mesencephalon, while the cortical cells may produce rhythmic impulses numbering twelve or thirteen per second.

J. von Kries, in a study of volitional muscular activity,⁵ calls attention to familiar voluntary rhythmical movements, and the number that can be made per second. For instance, in the repetition of la-la-la the muscles engaged in articulation and moving the jaw cannot be made to exceed in fre-

³ *Ibid.*, p. 512.

⁴ *Journal of Physiology*, 1885, pages 96 and 111.

⁵ *Archiv fur Physiologie*, 1886.

quency of contraction 6.2 per second. The fingers of a piano player are required to strike 11.2 tones per second in Chopin's Etude, op. 25, No. 2, and 12.3 per second in Czerny's *Schule der Fingerfertigkeit*, No. 1. Von Kries thinks that even this speed may be surpassed in short passages. The fastest voluntary movements of the wrist are about eight per second. The octave study, No. 8, in Chopin's opus 25, requires seven per second, while in the scherzo movement of Schumann's *Clavier quartette* the left hand must strike the keys rhythmically at the rate of eight times per second. Von Kries thinks that many virtuosos attain to a frequency of eleven per second. He used an instrument in his studies fashioned upon Marey's sphygmograph.

If we turn now to pathological conditions we find that the clonic spasms of epilepsy have a rhythm of ten or less per second; and the ankle clonus may be easily determined to be at the rate of ten per second or less. Some of my tracings of ankle clonus are six per second. The movements in nystagmus are very variable, but are usually from one to three or four per second, occasionally too rapid for counting, as the application of any myographic apparatus to the eye is almost impossible.

Marie⁶ examined the tremors of paralysis agitans and Basedow's disease. He found the rate of vibration five per second in the former disease, and eight to nine per second in exophthalmic goitre.

Charcot⁷ pictures two myographic curves, one of multiple cerebro-spinal sclerosis and one from paralysis agitans. They are of no particular value, being half diagrammatic. He found the contractions of paralysis agitans to be four to five per second. Ewald⁸ counted the oscillations also at five per second in this disease.

Grashey⁹ made use of Marey's sphygmograph in the study of the rhythm of oscillation in four cases of paralysis agitans. He made tracings of the tremor of the right and

⁶ *Contributions à l'étude et au diagnostic des formes frustes de la maladie de Basedow.*

⁷ *Maladies du système nerveux.*

⁸ *Berliner Klin Wochenschrift*, 1883, No. 32.

⁹ *Archiv für Psychiatrie*, Bd. xvi, S. 857, 1885.

left hands and of the tongue. By this means he fixed the number of vibrations from 4.14 to 5.34 per second.

Huber,¹⁰ by the application of Marey's sphygmograph to the bellies of the different muscles of the two arms, found that the number of oscillations varied from 3.43 to 5.57 per second. He also discovered that the rate of contraction varied in different muscles of the same person and in the same muscles on different days.

Gowers¹¹ shows some myographic tracings of tremor following hemiplegia, in paralysis agitans, insular sclerosis, general paresis, and hysteria in his new book. He makes the rate of oscillation in Parkinson's disease 4.8 to 7 per second, which is greatly in excess of the figures given by any other observer. Like Huber, he found considerable variation in different parts of the same person.

In my own studies of tremors I have also made use of a sphygmograph. I long ago abandoned the Marey sphygmograph for taking the pulse, and for three or four years have employed that manufactured by Edwards. Marey's was one of the earliest instruments invented, and the mechanical devices introduced between the pulse and the tracings are so crude and so lacking in delicacy that its use is open to many sources of error.

The Edwards sphygmograph (Fig. 1) possesses the highest qualities of mechanical precision and sensitiveness. The tracing needle and its adjustment are all in jeweled bearings and conical pivots with end jewels. It can be focused like a microscope, so to say, upon the pulse, by means of the milled nut on the barrel, and the vibrating blood column is brought as nearly as mechanical art can bring it into relation with the point of the tracing needle. Its sensitiveness is such that it may be easily applied to the tracing of fine oscillations of the tongue or fibrillary tremor in the lips. With this instrument I have been able to obtain beautiful transfers of muscular movements to paper. The myograms I present herewith will not exhibit the per-

¹⁰ *Virchow's Archiv*, Bd. cviii, S. 45, *Myographische Studien bei Paralysis agitans*.

¹¹ *Diseases of the Nervous System*, 1888, p. 1001.

fection perhaps that instruments especially designed for the purpose might do; yet much can be learned from them as to the innervation rhythm and irregularities of various muscular tremors.

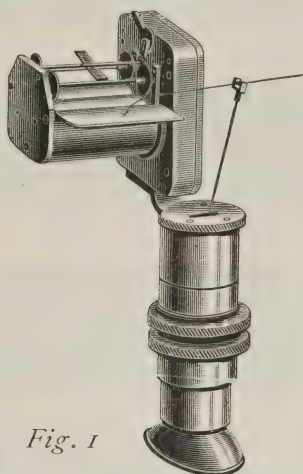
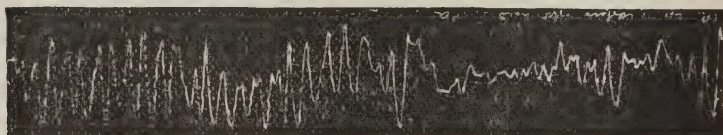
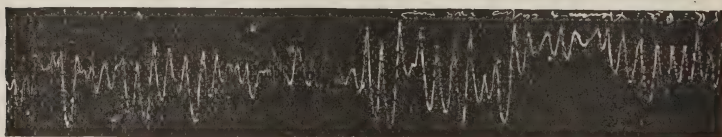
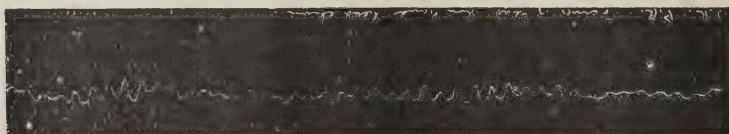
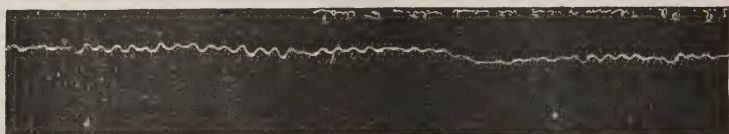
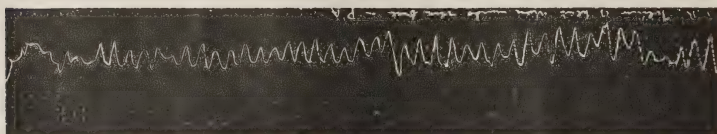
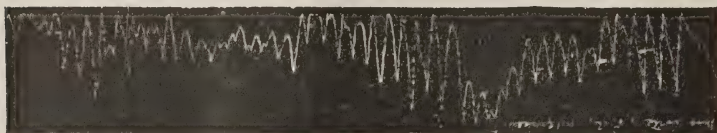
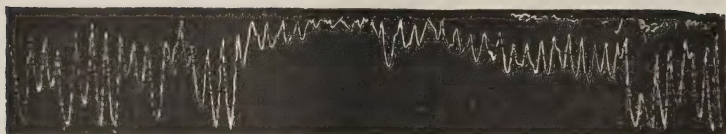


Fig. 1

In taking such tracings it will be found that the will of the individual upon whom the instrument is used greatly modifies the character of the vibrations. This must be expected, because we make, as is well known, a great distinction between tremors as regards this particular feature. We speak of the intention tremor of multiple sclerosis, where voluntary effort gives rise to the muscular oscillations, while in paralysis agitans, for instance, any volitional impulse usually suffices to momentarily interrupt vibration. Hence, in the application of the instrument to muscles exhibiting an intention tremor, any variation in the voluntary attempt to hold the member steady may either diminish or increase the extent of excursion. In paralysis agitans the patient is told when the instrument is in position to allow the limb to lie in perfect repose, but he is very apt to make an involuntary use of his will, if such an expression be permissible, and thus cause a momentary cessation of the tremor, or at least a decrease in the distance of oscillation. In such a series of muscular contractions as that of ankle clonus, the will is powerless to affect the motion of

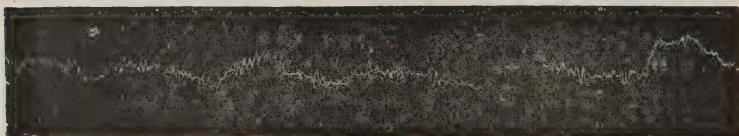
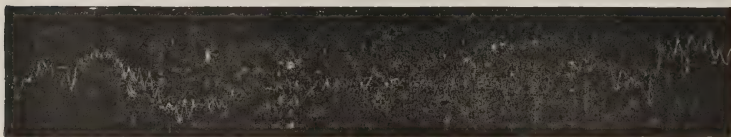
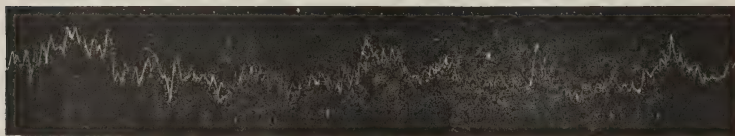
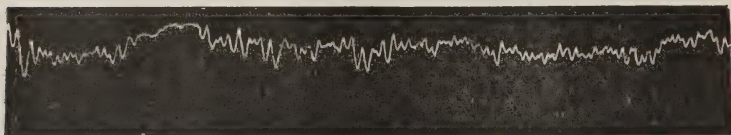
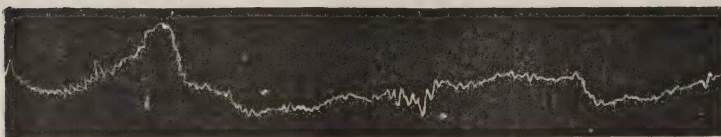
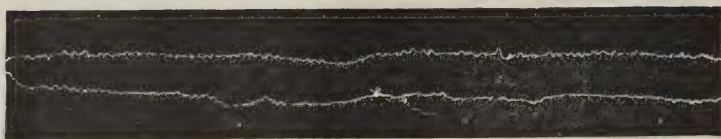
TEN SECONDS.



TREMOR OF PARALYSIS AGITANS.

- | | | | |
|----|--|-----------|-----------------|
| 1. | Tremor of extensors of carpus of right hand, | - - - - - | 5.3 per second. |
| 2. | " " " " | - - - - - | 5.1 " |
| 3. | Tremor of head while hands held a chair, | - - - - - | 4.4 " |
| 4. | " " " " | - - - - - | 4.6 " |
| 5. | Tremor of head, no effort with hands to keep steady, | - - - - - | 4.8 " |
| 6. | Tremor of Interossei, Case No. II, | - - - - - | 4.5 " |
| 7. | " " " " | - - - - - | 4.9 " |

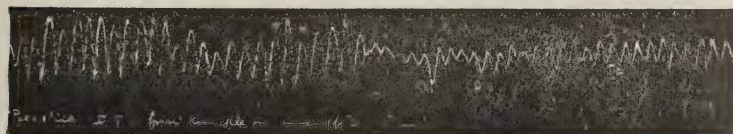
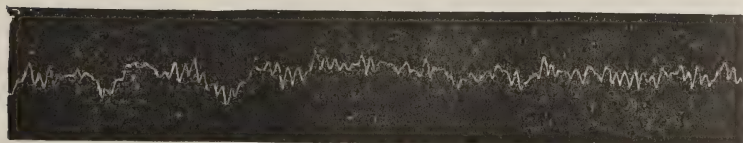
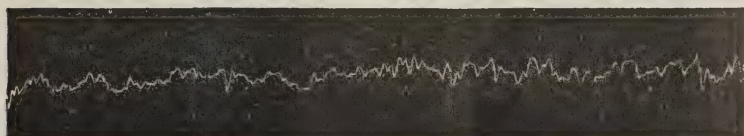
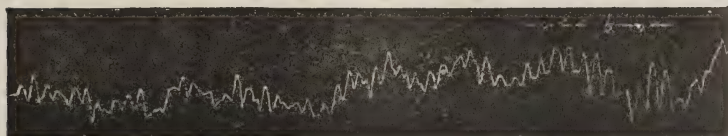
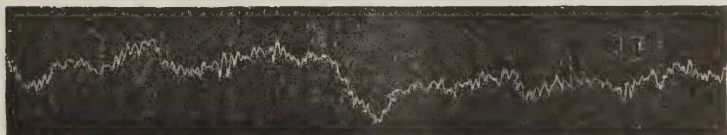
TEN SECONDS.



TREMOR OF MORBUS BASEDOWII.

1.	Taken from unsupported hand—wrists steadied against table,	12	per second.
2.	“ “ “ “ “ “ “ “	8.7	“
3.	“ “ “ “ “ “ “ “	9.9	“
4.	“ “ “ “ “ “ “ “	10	“
5.	Taken from wrist, right side, hands at rest on table,	9.7	“
6.	“ “ “ “ “ “ “ “	11.3	“

TEN SECONDS.



A COMPARATIVE SERIES OF MYOGRAMS OF VARIOUS TREMORS.

1.	Paralysis Agitans,	-	-	-	4.7 per second.
2.	Morbus Basedowii,	-	-	-	11.7 "
3.	Multiple Sclerosis,	-	-	-	5.4 "
4.	Hysterical Tremor,	-	-	-	7.7 "
5.	Neurasthenic Tremor,	-	-	-	7.4 "
6.	Delirium Tremens,	-	-	-	5.6 "

the gastrocnemius; unless the position of the foot be changed by the use of other muscles. My myograms of ankle clonus consequently are a strong contrast to all the others in point of symmetry of wave-length.

The tracings here presented all passed through the sphygmograph in ten seconds, and accordingly the waves of vibration may be counted and the rate per second determined. As did Huber with Marey's instrument, I found the rate of vibration in paralysis agitans variable in different parts of the body of the same individual. My determination of the average rate in this disease agrees with that of all other investigators (except Gowers), viz., 3.7 to 5.6 per second. The rate for the tremor of the head in one individual was 3.7 per second one day, while a few weeks later it varied from 4.4 to 4.8 per second. These differences are due in part to the variable volitional impulses, and also in part to a variable innervation rhythm, making the excursions greater at some moments than at others. As a rule, when the muscular oscillations are coarser there will be fewer per second. The matter of the existence of tremor in the muscles of the head and neck in paralysis agitans, once denied by Charcot, receives now general confirmation I think by most observers.¹² In my tracings will be seen many differences in the rate of tremor in Parkinson's disease, where taken from the carpal extensors, from the head, from the interossei, or from the supinators and pronators. There is a manifest lack of that uniformity which is generally ascribed to this tremor, but the reasons for this have already been discussed. At the same time

¹² I have seen five or six cases with undoubted tremor of the head, one where the tremor of the head was much greater than that of the arms. For cases of head tremor in Parkinson's disease, see the following authorities:

Oppolzer, *Spital Zeitung*, No. 17, 18, 1861.

Clement, *Lyon Medical*, No. 26, 1869.

Jones, *British Med. Journal*, 1873.

Westphal, *Charité Annalen*, iii. u. iv. Jahrg.

Demange, *Revue d. Med.*, ii., 1882.

Buzzard, *Clinical Lectures on Dis. of the Nerv. Syst.*, 1882.

Huber, *locus cit.*

Gowers, *locus cit.* (8 out of 37 cases).

the movement is seen to be more rhythmical than that of multiple sclerosis, ordinary alcoholism, hysteria, neurasthenia or morbus Basedowii. In delirium tremens, however, my tracings are quite as regular as in paralysis agitans. It must be borne in mind that myograms are subject to the same modifications in character as are sphygmograms from the variable pressure brought to bear by the holder of the instrument upon the moving muscle or artery. Indeed, there is possibly more modification from this source in using the instrument for the record of muscular tremors, because some of the tremors develop only when the fingers or hand are unsupported. The operator can fairly remedy this, as far as his own steadiness in holding the instrument is concerned, by supporting his elbows or arms upon a table and being seated. The arms of the patient may be steadied against the table, but even then his respiratory movements may affect the myogram somewhat, because his arms are attached to the moving thorax. A real respiratory curve may be seen in some of my myograms, notably those of the tremor of exophthalmic goitre. None of these difficulties, however, alter the rate of rhythm and will not change other peculiar characteristics if reasonable care be taken.

The tremor of paralysis agitans is almost the only one, with the exception of shivering from cold or terror, developed when the body is in a state of rest. Nearly all the others belong to the category of intention tremors, or to a class originated when limbs are outstretched without support. I am not so sure but that the last-named are also intention tremors, since voluntary effort is required to extend a member. The number of oscillations in well-developed multiple sclerosis I find to be 4.6 to 6.3 per second, but in another case where the tremor is just beginning to be perceptible, the innervation rhythm is numerically greater, viz., 7.9 to 8.1 per second. In a case presenting symptoms common to both paralysis agitans and multiple sclerosis, I made a diagnosis of the latter disease upon the fact that the rate of vibration was almost twice as great as it would be in paralysis agitans. The tremor was slight

and an intention tremor in character to be sure, but cases of Parkinson's disease have also been cited with an intention tremor (Gowers). The great irregularity of oscillation in multiple sclerosis is well demonstrated in some of my myograms.

The tremor of morbus Basedowii is fine, irregular, and has an innervation rhythm of 8.7 to 12 per second, according to my observation. Marie, as already mentioned, found it 8 to 9 per second.

Hysterical tremor is also fine, lacking in uniformity of excursion, and rapid, attaining a rate by my measurement of 7.6 to 7.8 per second.

The tremor developed by alcoholic intoxication is very variable. In ordinary alcoholism it presents the characteristics of that in hysteria and exophthalmic goitre as regards fineness, irregularity and rate of motion. I determine the rate to be 8.5 to 11.2 per second, varying within that limit in different persons and in the same persons at different times. Delirium tremens, however, presents a slower rate, 5.6 to 6.8 per second in my investigation, and somewhat greater uniformity of movement.

The few myograms I have taken in neurasthenia have the character of the tremors of ordinary alcoholism, Basedow's disease and hysteria. The frequency is 7.4 or more per second.

If we compare the rates of some of these tremors with the normal innervation rhythm of muscle as determined by the experiments of Horsley and Schäfer already mentioned, and by Beaunis,¹³ about 10 per second, we note at once the near coincidence of the more rapid tremors with the normal rate, and the fact that the slower oscillations of paralysis agitans, multiple sclerosis and delirium tremens are about half that rate. It would seem that just as ten impulses along a motor nerve combine to make one normal continuous muscular contraction, so under pathological conditions the impulses may affect the muscles singly or in fused groups of two or more to produce rapid or slow tremors.

¹³ *Physiologie humaine*, 1876, page 273.

Recently Wolfenden and Williams,¹⁴ with a special myographic apparatus, have demonstrated the dicrotic character of the oscillations in paralysis agitans and to some extent in disseminated sclerosis. They found the rate in morbus Basedowii to be 10.8 to 11.5, in paralysis agitans 5.1, in disseminated sclerosis 5.8, and in a case of lateral sclerosis 5.5 per second, figures which correspond closely with my own.

As regards the origin of these tremors, the usual hemiplegic progress of paralysis agitans, occasional inequality of pupils, the cessation of movement, as a rule, during sleep and other arguments, lead us to suppose a genesis of the vibratory contraction in the cerebral cortex. The tremor of fear is without doubt of cortical origin. That of multiple sclerosis is best explained as also developed from the motor areas of the brain, its jerky character being ascribed by Charcot and Gowers to resistance to motor conduction at sclerotic foci, and by Stephan¹⁵ to resistance by sclerotic changes in the optic thalamus. With at least equal justice it seems to me we may surmise that all other tremors, save those fibrillary in character, must be generated by intermittent motor impulses from the gray matter of some portion of the central nervous system. However, until all the physiological facts are accurately determined, the whole matter of the finer pathological movements must remain obscure.

This contribution of mine to the study of tremors is rather in the nature of a preliminary report upon investigations in this direction. While there may not be sufficient data here to arouse any great hopefulness of the diagnostic value of this means of examination, still considerable light may be thrown upon the nature of tremor in general by the employment of this method, and it demonstrates at any rate that we have a convenient and delicate instrument at hand for recording vibratory muscular movements for future comparison and study.

¹⁴ British Med. Journal, May 19, 1888.

¹⁵ *Archiv. f. Psychiatrie*, Bd. xviii., S. 734, and Bd. xix. S. 18.

DISCUSSION.

Dr. SINKLER asked whether the author had obtained the knee clonus and the ancle clonus at the same time.

Dr. PETERSEN replied that he had never attempted it.

Dr. SINKLER explained that if any one first started the patellar clonus and then the ankle clonus, usually the latter would stop the former ; but that sometimes both would continue for a few seconds together. It would be interesting to know whether they coincided.

Dr. HARE had had some experience in attempting the registry of tremors. The practical difficulty lay in obtaining a tracing of the up and down and the transverse movement at the same time. His apparatus had consisted of a vertical piece, a short arm with a hole in the end, through which was a reed like the rib of an umbrella two feet long and carrying a pen at a right angle at one end. The other end of the rod was attached to the sole of the foot, and by its position the image could be obtained magnified or the reverse. The recording sheet was eight feet long, traveling at considerable speed. Some tremors were so rapid that they required a rapid registering surface in order to catch them. Again, a tremor would have a certain character at one time and a different character at another. A registering surface of only ten inches would often give an erroneous result.

METHOD OF PREPARING BRAINS AND OTHER ORGANS FOR ANATOMICAL AND PATHO- LOGICAL DEMONSTRATION.

By I. W. BLACKBURN, M.D.,

SPECIAL PATHOLOGIST TO GOVT. HOSPITAL FOR THE INSANE, WASHINGTON, D. C.

THE material used for the purpose is the so-called "Japan wax," which is really a concrete oil, the product of *rhus succedanea*, Ln., a tree of Japan. It comes in large rectangular blocks about one and a half inches thick ; has a yellowish white color, and a somewhat rancid smell and taste. Its melting point varies from 107° F. to 131° F., and at ordinary temperature it is firm and solid. It is insoluble in water, scarcely soluble in cold alcohol, slightly so in boiling absolute alcohol, ether, and in turpentine. It is very soluble in *chloroform*, *benzole*, and *xylol*.

The specimen or organ, in this case the brain, is carefully hardened in some reagent which will preserve its size and shape as perfectly as possible, and the best for this purpose are Müller's fluid and Erlicki's solution.

Other hardening agents, such as alcohol, chloride of zinc and nitric acid, shrink the tissue too much, though the color is, perhaps, more pleasing. Hardening may be hastened by injecting the fluid into the vessels before removing the membranes, and these may be removed just as well after three or four days' immersion in the fluid.

After hardening for about five weeks in Müller's or a shorter time in Erlicki's fluid, the specimen is removed, washed, placed in dilute alcohol, and gradually advanced through alcohols increasing in strength until absolute alcohol is used. When thoroughly dehydrated by the use of absolute alcohol it is placed in a saturated solution of Japan wax in *chloroform*, and allowed to remain until the alcohol

is displaced by the chloroformic solution.¹ The organ is then transferred to a bath of melted wax and kept therein at the melting point until thoroughly infiltrated. After infiltration is complete, the specimen is removed from the bath, the wax drains from the surface leaving it smooth, and when cool it may be varnished if desired, and upon the varnish, painted or lettered to suit the purpose of the operator.

If the wax cannot be kept melted continuously during the process of infiltration it is better to lift out the specimen and replace it in the chloroformic bath, as when cooled in large masses the wax has a tendency to crack, and by this the preparation might be injured. A small proportion of paraffine, with which the wax is perfectly miscible, will prevent cracking and in no way interfere with the process.

Specimens hardened in the chromic acid salts and prepared by this method have a dark olive or bronze color; those hardened in alcohol or chloride of zinc become slightly darkened by the process. There is no odor to the specimens except that of the wax, which is not disagreeable.

The preparations are permanent in the air, are more durable than wax models, and the shape and size are perfectly preserved. Specimens prepared two years ago show no appreciable change.

The time required for the various steps of the process cannot be definitely given, as it will depend upon the size and character of the specimen; but after thorough dehydration, which is the most tedious part of the process, a hemisphere should be allowed to remain at least three days in each bath.²

¹ Chloroform is preferred to the other solvents on account of its safety; the others are highly inflammable.

² For the use of the "Japan wax" in microscopy, see article in the Transactions of Ninth International Medical Congress, vol. iii., page 407.

CASE OF PROBABLE TUMOR OF THE PONS.¹

By MARY PUTNAM JACOBI, M.D.

THE child whom I have the honor of bringing before the Society to-night has the following history.

Jennie Baer, aged ten years. The morbid symptoms are said to date from two and a half years ago, when the child was frightened by a trivial incident that she remembered with rather remarkable tenacity, and describes with lively interest. She was fastening the apron of a school friend, when the mother of the latter ran at her, uttering some injurious epithet. The child, the patient, ran away as fast as she could, up several flights of stairs to her own room, and immediately began to tremble. Two days later, on going to school, this trembling was noticed by the teacher. It continued, and even increased for two weeks, at the end of which time the teacher advised removal from school. She has never returned, nor attempted any systematic exertion since, bodily or mental. A little while after the removal from school, the parents noticed that the child walked as if intoxicated. The father imitates this mode of walking by slightly swaying the body from side to side, leaning a little forward, dragging one leg after another without lifting them from the floor, but somewhat swinging each in a semicircle, as if to enlarge the base of support. It is the walk "en fanchant" of the French writers.

At this time, and on account of the trembling and inability to use the hands for writing or other fine work, the parents consulted Dr. Lilienthal, who diagnosed St. Vitus' dance, and treated the child accordingly, but without benefit. At this time, though there was said to be twitchings of the limbs, there was none of the muscles of the face. The head was, however, habitually drawn a little to the left side, and the child exhibited the taciturnity common in chorea.

¹ Read before the Neurological Society, December 2, 1888.

After this period, *i. e.*, a month or two from the debut of the accidents, the child seemed constantly tired, and usually spent the time lying down. She played very little.

At the end of about three months the child began to have trouble with walking, would frequently stumble, and occasionally fall. Could not go up and down stairs readily. It was at the same time that she began to grow very fat.

She was brought to my clinic at the Woman's Medical College, in May of this year, 1888, after two years' continuance of the above symptoms. She had then been suffering during several weeks from headache, continuous, diffused, but by no means violent. It may be noted at this point, that this headache disappeared after a duration of seven or eight weeks, and has not returned. There had been two attacks of vomiting, at an interval of several weeks. These have not been repeated.

When the child was seen by me in May, I was at once struck by her large fleshy size, the unusual development of the limbs, and especially the great size of her head. The measurements taken at the clinic have been lost; but a month later, Dr. Petersen kindly made a careful examination of the head for me, and, together with some other determinations, the following measurements were taken:

Circumference equals $57\frac{1}{3}$ centim., or $22\frac{4}{5}$ inches.

Naso-occipital line equals 38 centimetres, or 15 inches.

Binauricular line equals 38 centimetres, or 15 inches.

I omit for the moment the other calculations made by Dr. Petersen.

I could not ascertain whether the mother had been impressed by the large size of the child's head, or whether she had noticed that it increased. The habitual expression of the child's face was apathetic; the eyes heavy and lids drooping; but if spoken to, her face lighted up and she replied intelligently and with a smile.

There was no hyperæsthesia of the scalp, and percussion of the head elicited no pain. There was no disturbance of sensibility of any kind in any part of the face, trunk or limbs. No sign of paralysis of any cranial nerve. The movements of the upper extremities were normal, though

there was a little clumsiness in the finer movements of the fingers, as in picking up a pin. There seemed to be some paresis of the trunk muscles, at all events of the extensors of the spinal column; for the child avoided sitting upright, and continually leaned against the back of the chair for support. The conspicuous disturbance of function was in the lower extremities. The child could, though with some difficulty, rise from her chair and stand without support, but only for a minute or two, for she then fell forward. She could walk a few steps across the room, but then also would fall forward unless supported. During the attempt at walking, the body swayed a little from side to side, and the legs described the semicircular curves already mentioned. A month later it was noticed that the right ankle bent while walking, so that the foot turned inward. This was not perceived in May. At that time the child could walk up stairs more easily than on a level, because she supported herself by the bannisters. Faradaic sensibility and motility were intact for all muscles.

The knee-jerk was somewhat exaggerated on both sides.

The visceral functions were all normal; the appetite was even excessive. There was no unnatural drowsiness.

A re-examination was made of the case on October 13th.

The condition was found much aggravated in many respects. The circumference of the head has somewhat enlarged a little. Having been 57 centimetres, or $22\frac{3}{8}$ inches, when measured by Dr. Petersen in June, it is now $23\frac{1}{4}$ inches, a difference of $\frac{1}{2}\frac{3}{8}$ of an inch. The naso-occipital and binauricular measurements remain the same.

The general appearance of the child is as already described; but she has grown still fatter, the girth of trunk, as measured by her dress which has burst out, being increased at least three inches.

The intelligence remains clear, notwithstanding the habitual listlessness. The child describes certain lines drawn on paper as vertical or horizontal, though she has not heard these terms since she left school two and a half years ago. She also relates the story of her original fright,

and inquires with interest concerning the prognosis of her disease.

A new symptom now exists, in the intermittent divergent strabismus of the right eye. This is occasionally quite marked, but can always be overcome by voluntary effort, when converging the eyes to look at an object placed not too near. At a distance of two feet the nature and number of the objects are accurately distinguished; but when five lines are drawn on a paper, and the child looks closely at them, she calls them four. If she attempts to count the lines by placing her fingertip on them one after the other, the difficulty is increased. In six trials she invariably skipped the third line, and could not accurately touch the fifth. (They were drawn horizontally one above the other.) This difficulty depended partly on the inability to move the finger with precision; for it was agitated with slight choreiform movements during the intended act, which delayed its accomplishment, as is the case in sclerosis. But, in addition, there seemed to be some visual defect which interfered with the exact guidance of the finger and which rendered the patient unconscious of the fact that the finger had been placed above the line instead of on it. I could detect no diplopia; but I believe the lines seemed blurred to the child during the effort of convergence of vision upon very near and similar objects. This blurring she could not be made to describe.

There is no evidence of paralysis of the facial. The tongue protrudes perfectly straight. The uvula is slightly deviated to the right side. The tonsils are much enlarged.

Thus the only morbid symptoms in the sphere of the cranial nerves, apart from the optic, are: 1st, the intermittent irritation of the right abducens, as indicated by the intermittent divergent squint; 2d, the difficulty of converging the eyes upon near objects, so as to see them without blurring—difficulty apparently due to the relative or positive weakness of the right internal rectus.

The ophthalmoscopic examination was made later.

Upper Extremities.—The arms are very large; the forearms do not seem to be disproportionate to the age of the

child. All movements of the arms and forearms can be executed without perceptible difficulty. Difficulty first appears when the child attempts to carry a glass of water to her mouth. Left to herself, she takes this in both hands, as if distrustful of her ability to hold it in one, although, on being tested, she can do so, and even, though unsteadily, carry it to her lips. When the glass is held by both hands against the mouth, she drinks readily until it is half emptied, but then seems unable to tip the glass at a greater angle in order to drain it. This movement necessitates the inclination of the hands on the radial border of the forearms, action effected by the supinator longus, and the longer and shorter radial extensors of the wrist. When there is no weight in the hand, the child can flex and deflect the hand on the radial border of the wrist; the difficulty only occurs when the hand is carrying a weight and for a special purpose. There is therefore no actual paralysis of the muscles, but paresis and diminished power of co-ordination.

A similar difficulty of co-ordination is shown in the fingers when the child tries to write. Before the hand reaches the pen it is agitated with slight choreiform or ataxic movements, as is also the case when she picks up a pin. She cannot go straight to the object. She places her head to one side, the right, in order to guide her hand in writing. She then forms the letters very imperfectly, and cannot write them in a straight line. The word (she resigns the attempt after the first word) always runs obliquely off the page, running up sharply from left to right. It is to be remembered, in connection with this test, that the child's education ceased when she was only eight years old, and before she had really learned how to write. Apart from efforts at functional co-ordination, all movements of the fingers can be performed voluntarily and without distinctly perceptible diminution of force. The grasp of the hand is normal for a child of her age. Sensibility is intact for the whole upper extremity.

The faradaic contractility and sensibility are intact.

Trunk.—The child cannot support herself upright, even in a sitting position, for more than a minute or two, but

leans back in the chair, showing paresis of the spinal extensor muscles.

The thighs and legs are noticeable for their large size, although the apparent muscular enlargement of the four extremities is not as striking as is that of the thorax and abdomen. While sitting, the child can extend the legs on the thighs perfectly, but not vigorously, and can flex, extend, and adduct the feet and toes. Abduction is extremely feeble on both sides. Nevertheless, she is unable to support herself standing for more than a half minute, and cannot now walk at all unless supported. With support she walks, dragging and to some extent swinging round the legs, while both ankles bend under her, bringing the feet into exaggerated varus. Thus there has been marked deterioration in the functional power of the limbs since May, and even since June, when the special weakness of the peroneal was first observed, and then only on the right side.

There is no retraction of the gastrocnemii muscles. The child has never walked on the toes, nor never exhibited the special phenomenon of pushing back the thighs or of climbing up them in rising. Nor was there ever lordosis, but the trunk always fell forward.

In May, as already stated, the child did pull herself up stairs by the aid of her arms. Also, while she was yet able to walk unsupported, the body did sway from one side to the other, as if from alternate inclination of the pelvis, and the legs spread outwards to enlarge the base of support. These symptoms have now disappeared, as the paresis has increased so far that the child cannot even attempt to walk unsupported.

The absence of retraction in the gastrocnemii is paralleled by similar conditions throughout the limbs, where neither contracture nor deformity exists. The sensibility is intact. All the muscles contract to the faradaic current, but the peroneal muscles demand a stronger current than the others. Thus the quadriceps extensors contract at 300 millimetres of the induction coil, the peroneal not under 260. The knee-jerk is moderately exaggerated. No ankle clonus can be obtained.

The limbs are constantly cold, subjectively and to palpation.

In addition to the foregoing symptoms may be noted :

That the child suffers no pain in any part of the body, nor has ever done so except for a moderate headache in last spring.

There is neither nausea, vomiting, nor anorexia, but, on the contrary, an excessive appetite, bordering on bulimia. There is occasional sighing respiration.

There is no constipation, but a condition of the bowels approaching incontinence, inasmuch as the child is unable to restrain the impulse for evacuation when this makes itself felt; and the impulse always occurs after any mental excitement, as if she has been laughing. The control of the bladder is similarly imperfect.

On the 28th of October Dr. C. S. Bull kindly made an ophthalmoscopic examination of the child. He found choked disk in both eyes, outlines much blurred, veins swollen and tortuous, arteries narrowed and in some places disappeared; process beginning to recede, but leaving an atrophy of nerves, which is now incipient, but tending to increase. Acuity of vision much diminished, only $\frac{1}{100}$. Spasm of right abducens. He diagnosed a descending inflammatory process of the optic nerve.

In 1887 Dr. Hun, of Albany, presented the following case, completed by the autopsy, to the American Neurological Association :

Gliomatous hypertrophy of the pons.

Female, æt. 6; father died a little more than a year after she was born, with symptoms of melancholia and dementia.

For several years left leg has tired easily; left foot shows a tendency to turn in; wore rubber straps; otherwise excellent health until two months ago, when attack of croup and cough. Pain felt in head with each cough. Three weeks before coming under observation the patient began to walk badly, and seemed to have trouble in balancing herself while walking. She had an excessive appetite and vomited a little at times.

When first seen the patient was a well-nourished, intel-

ligent girl, but with a vacant expression. Her speech was drawling. Her head was drawn towards the right shoulder most of the time, especially if she made any exertion. She stood with her feet wide apart, and was careful not to lose her balance. In walking, her right leg was more rigid than her left, so that she took freer and longer steps with her left leg, and therefore in walking tended to go in a circle, turning always to the right. Her walk resembled that of a drunken person. The movements of the arms, especially of the right, were very awkward, but she held them in no fixed position.

There was no disturbance of sensibility in any part of the face, body, or extremities, and she recognized objects placed in her hands when her eyes were shut.

The plantar reflexes were normal. The knee-jerk was exaggerated, especially on the right side. There was no ankle clonus.

On ophthalmoscopic examination, well marked optic neuritis was found in both eyes. Urine contained neither albumen nor sugar.

The patient was first seen on the 17th of April, and from this time the symptoms rapidly increase, passing through gradations I need not here describe, until a fatal termination was reached on the 13th of June.

At the autopsy, the pons varolii was found enlarged to three or four times its normal size, and on section, it was found to have been replaced by a tumor, apparently a glioma, which so well preserved the normal appearance of the part that it looked like a greatly hypertrophied pons.

On microscopical examination, the nervous elements of the pons were found to be encroached upon, infiltrated, and in some places destroyed by a great accumulation of small cells. The proliferation of cells was not limited to the pons, but extended throughout the crura cerebri and medulla, and especially in the roof of the aqueductus Sylvii. The prevailing character of the cells was spindle, but there was a considerable number of spheroidal cells, both large and small.

The bones of the skull were thin. The subarachnoid

fluid increased. The cerebral convolutions flattened, and the cerebral substance very œdematous. Lateral ventricles greatly dilated, and filled with fluid of normal appearance.

Dr. Hun ascribes the inco-ordination of movements to pressure upon the transverse fibres of the pons, and the origin of the crura cerebelli; the absence of absolute paralysis to the fact that the nerve elements were compressed but not destroyed by the infiltration. He explains the absence of sensory disturbance by a greater resistance of sensory function, even when sensory fibres are submitted to the same pressure as motor fibres. He notes that the tumor in its growth produced no symptoms of irritation, that there were no convulsions, and but little headache.

In all these circumstances, the case closely resembles the one I now present. Other points of resemblance are the long duration of slight muscular inco-ordination, and even paresis of the lower extremities, without other symptoms, up to a certain date; then the rapid deterioration in a few months; the fact that the most marked paresis was shown by the turning in of the right ankle; the exaggeration of the knee-jerk, the other reflexes remaining normal; the vacant expression of face, and apathetic appearance, although the memory and intelligence were entirely preserved; the indistinctness of articulation; the optic neuritis, the slight and transitory squint, which in Dr. Hun's case was convergent, in this one divergent and unilateral, without paralysis of any ocular muscle; finally, the excessive appetite and excellent preservation of nutrition. The nutrition in our case is even exaggerated, so as at one time to have suggested a general lipomatosis.

The optic neuritis is, as well known, a most important indication of intra cerebral tumor; and indeed I did not permit myself to make a positive diagnosis until the existence of this symptom had been established. Bramwell enumerates the following diseases other than mental in

¹ A case of tumor of the pons and medulla in a child of two years, related by Hobson, (*Brain*, Vol. IV., p. 531), differed greatly from ours, especially in the number of cerebral nerves involved.

which double optic neuritis may occur. It is nearly always present in lead encephalopathy; it is not uncommon in meningitis and cerebral abscess; and it may occur in Bright's disease. It has occasionally been associated with uterine derangements, hypermetropia and anæmia, which, when complicated with hysterical symptoms, may be easily mistaken for cases of intra cranial tumor.

In our case all the foregoing conditions may be readily excluded.

Stephen Mackenzie has published in the second volume of *Brain*, a case where an optic neuritis depended upon a diffused cerebritis, resulting in general atrophy of the brain, and an inflammation extending down the optic nerve. The symptoms of this remarkable case, which has often been quoted, are chiefly to be referred to the cortex of the brain, and in no wise resembled those of this child.

When I first saw the patient, the unusually large size of the head, the apathetic expression of face, the moderate degree of motor disturbance, the diffused nature of the symptoms, led me to suspect an hypertrophy of the brain, due to diffuse lobar sclerosis. "In such cases," observes Gowers, "the symptoms have been very similar to those of cerebral tumor, headache, vomiting, local palsy, convulsions."³

"The symptoms," observes Schmidt,⁴ "of diffused sclerosis of the brain are gradually increasing muscular weakness, manifesting itself especially in the lower extremities, giving rise to an unsteady, stumbling gait and frequent falling. There are also epileptic spasms, constant or intermittent attacks of headache, vertigo, tinnitus aurium, photophobia, dimness of sight with dilatation of the pupil, blunting general sensation without anæsthesia. The skull may be enlarged and thinned."

So Richardière⁵ affirms: "Convulsions and attacks of tremor and muscular rigidity are never absent in cases

³ Diseases nervous system.

⁴ Peppers Archives of Medicine.

⁵ Scleroses encephaliques primitives de l'enfance. Paris, 1885.

of diffused sclerosis." Both these symptoms have been entirely lacking in the case of Jennie Baer. On this account the hypothesis of the diffuse sclerosis was finally rejected.

To sum up: the reasons for diagnosing an intracranial tumor in the case of the child before you, are: 1st. A diffused motor disturbance, beginning as inco-ordination and difficulty of equilibrium, increasing at first slowly, then with sudden rapidity to such paresis of the lower extremities as renders station impossible; of the trunk muscles, as renders upright sitting difficult; of the upper extremities, as interferes with the more delicate movements of the hands. 2d. Preservation of faradaic contractility, absence of nutritive lesion of the paretic muscles. 3d. Gradual enlargement of the head. 4th. Moderate apathy of expression and dulling of intelligence. 5th. Spasm of right external rectus. 6th. Double optic neuritis, commencing atrophy of the optic nerves, marked diminution of visual acuity.

The circumstances which indicate a localization of the tumor in the pons are: 1st. The absence of convulsions, a negative fact of great importance, and frequently observed in slowly infiltrating tumors of the pons. 2d. Absence of marked or definite symptoms, and of monoplegic spasm or paralysis, tend to exclude tumors of the cortex. 3d. Absence of hemiplegia excludes the basal ganglia. 4th. The same, with absence of symptoms in the sphere of the motor oculi, excludes tumors of the crura or base of the brain. 5th. Absence of headache, nystagmus and vomiting, and the development of motor paralysis in addition to the original motor inco-ordination, excludes, I think, the cerebellum. 6th. The general march of the symptoms, the bilateral character of the paresis, the inco-ordination, even the absence of anæsthesia or pain, are precisely what have been observed in slow growing tumors of the pons. In seven out of thirty cases of pontine tumor tabulated by Bernhardt, no disturbance of sensibility existed but headache, and even this failed in two cases, as also in that of Dr. Hun. The irritation of the right abducens, the only cranial nerve at present affected except the optic, is in accordance with a localization of disease in the pons. The excessive appetite of the

child is a symptom probably to be referred to the medulla.

Characteristic symptoms of pontine tumors which are, as yet, absent, are: 1st. Alternate paralysis of face and limbs. 2d. Paralysis of the hypoglossus, or of any cranial



nerve, other than the optic. 3d. Marked difficulty of deglutition or articulation. Some defect of the latter function is, however, noticeable, and there is occasionally sighing respiration. The tumor could hardly be situated in the upper

region of the pons, or there would be motor oculi symptoms, or others referring to the corpora quadrigemina; while at the lowest portion of the pons, the spinal accessory nucleus or the hypoglossal nerve should be involved. We may



infer that the growth is situated about the middle region, that it is bilateral, that it is below and anterior to the nucleus of the fifth and anterior to the nucleus of the seventh nerve. That on this account the lower extremities

are paralyzed before the upper, and the seventh has so far escaped. We must further infer that the lesion consists of



a very gradual infiltration of elements, gliomatous or sarcomatous into the nerve tissue, and is not a sharply defined new growth, forcibly compressing any localized bundles of nerve fibres.

One other hypothesis remains,⁶ namely, that there is a tumor of the cerebellum which presses on the pons. The reasons which militate against this hypothesis are, that there have never existed any characteristic symptoms of cerebellar tumor, except the tottering walk and loss of equilibration. But these symptoms are also observed in the infiltrations of the pons which affect the transverse crura cerebelli.

In the four cases of cerebellar disease reported by Dr. Seguin,⁷ the following symptoms, positive and negative, were observed, differing from the history of Jenny Baer.

CASE I.—*Positive*: Headache, nystagmus, convulsion. *Negative*: Absence paralysis until after hæmorrhagic seizure two months before death.

CASE II.—*Positive*: Vomiting, violent headache, convulsions.

CASE III.—Diffused headache, vomiting, double exophthalmus, absence paralysis.

CASE IV.—Repeated nausea and vomiting, severe occipital headache, absence paralysis.

The paralysis in the case of Jenny Baer is not indeed complete, but it is sufficient in the lower extremities to render standing quite impossible, which is more extensive than is ever the case with purely cerebellar tumors.

⁶ This was strongly urged by Dr. Sachs at the meeting of the Neurological Society when the above communication was read.

⁷ Journal Nervous and Mental Disease, vol. xiv., April, 1887.

PERISCOPE.

THERAPEUTICS OF THE NERVOUS SYSTEM.

THE SUSPENSION TREATMENT OF LOCOMOTOR ATAXIA.
—Dr. Paul Blocq has recently read a paper at the Société Médico-Pratique (Rev. Gén. de Clinique, Feb. 14, 1889) on the results of Dr. Motchonkowsky's treatment of locomotor ataxia by suspension, as carried out for the last few months at Salpêtrière. The treatment was suggested to Dr. Motchonkowsky, of Odessa, by observing the benefit which an ataxic patient, also suffering from spinal curvature, derived from the suspension required in applying a plaster jacket. On suspending other ataxics in a similar manner, he found that very marked improvement in the lightning pains and the motor inco-ordination followed, and vesical and sexual power was restored.

At the Salpêtrière fifteen patients have been submitted to nine hundred suspensions since last October, with marked benefit in many cases. The most usual signs of improvement were the re-establishment of the sexual function, the disappearance of bladder troubles, diminution and disappearance of the lightning pains, with improvement in motor co-ordination, so that the patients who had only been able to walk with the help of an attendant one side and a staff on the other could leave the hospital after treatment without help of any kind.

Dr. Paul Blocq has also applied suspension with benefit in Friedreich's disease. The suspension was applied two or three times a week for periods varying from thirty seconds to three minutes each time. Improvement in walking began in the case of a girl, aged fourteen, in the second week of treatment. Later, a spoon could be carried to the mouth with the eyes closed, and she now learns the piano,

writes with little tremulousness, can walk better, can stand with the eyes closed, and the catamenia have become established. The tendon reflexes are, however, still wanting, and scansion and nystagmus remain.

As Professor Charcot remarked, these results in a disease which has always been slowly progressive and almost invariably fatal, are worthy of attention. He suggests that the suspension may act by modifying the circulation of the spinal cord, or by stretching the nerves as they leave it. Whatever its *modus operandi* may be, it is certain that suspension is an agent of considerable power, since serious accidents have occasionally happened during the application of a Sayre's jacket, and it is therefore to be used with discretion and care.

We understand, also, that a number of patients suffering from various forms of chronic degeneration of the nervous system are being treated by suspension in various London hospitals. It is, of course, too early to form any definite opinion of the value of this treatment; but, so far, the results have been encouraging. A patient at present in St. Mary's Hospital was "suspended" on January 22d, and at intervals from that day, by Dr. de Watteville, physician in charge of the electro-therapeutical department, who has reported the case as progressing satisfactorily. The most apparent improvement consists in the increase of gait and equilibration, as manifested by the ease with which the patient can turn around when ordered to do so. Dr. Althaus informs us that he has found it beneficial in two cases of tabes; lightning pains in the one case, and in the other gastric crises, have ceased. In a case of severe paralysis agitans the tremor ceased for thirty-six hours after the first suspension.—*British Medical Journal*, February 23, 1889.

The Berliner kl. Wochensch., No. 8, reports that the suspension treatment has been tried in the clinics of Professors Eulenberg and Mendel with equally favorable results, the suspensions, which took place three times a week, being at first of one minute's duration, increasing by half a minute up to three minutes. About twenty patients have thus been treated; and although, of course, the time is too short to

announce any positive results, two facts have been found to follow the treatment, viz.: 1. A certain number of patients have, immediately after the suspension, a readier and freer gait, less staggering, and complain less of lancinating pains (in some, also, improvement was noted in visual symptoms). 2. No ill effects have followed the practice. Our contemporary warns physicians and the public from hasty and exaggerated hopes in its efficacy.—*Med. News*, March 16, 1889.

A very fine exhibit was made at the American Institute by the Jerome Kidder Manufacturing Company, 820 Broadway, New York, makers of Dr. Kidder's electro-medical apparatus. The company may well take pride in their exceptionally fine line of instruments, medical batteries, etc. The company have received the highest awards from the Institute since 1872, and so far from falling off in their productions, manifest a steady onward and upward tendency. They show a full line of galvanic batteries, galvano-caustic batteries, many styles of faradic batteries for family and physicians' use, tip batteries, surgical instruments, cauteries, and special appliances for an endless variety of medical and surgical operations.—*Electrical World*.

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THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

A CASE OF HEREDITARY MUSCULAR
DYSTROPHY.¹

By JAMES J. PUTNAM, M. D.

THE patient whose case is to be reported is a woman forty-five years of age, in comfortable circumstances. She has always been in good general health, and has not been exposed to severe muscular fatigue. In brief, her early history offers no explanation of her present symptoms. She can, however, remember that even as a child she was unable to run and jump like other children.

When she was about seventeen years old the failure of muscular power became prominently noticeable, and it has slowly but steadily increased ever since.

Her condition at present is as follows :

She walks excessively slowly, with a waddling gait, using her legs as little more than crutches. When she throws her whole weight upon either foot, as in going up or down stairs, the foot turns so that the leg almost rests upon the ground, and she is wholly unable to go up or down stairs without either clinging with her hands to the balustrade, or going on all fours.

The hands and arms are still useful members, though feeble. When examined more closely, it is found that the

¹ This case was reported in connection with the paper upon "Muscular Dystrophies," read by Dr. Sachs at the last meeting of the Neurological Association.

movements of the feet and toes are practically though not absolutely abolished, but that all others are possible to a greater or less degree.

The thigh-trunk muscles are excessively feeble, as is shown by the manner in which the trunk is thrown from side to side when the patient walks. She can, however, sit erect, and stands without marked lordosis.

Her best movements are those of the shoulder, and the muscles there appear to be in the best condition. The muscles below the knees are excessively wasted, but rather dense, and with the skin drawn tightly over them.

The biceps of both sides are also much wasted, looking as if hollowed out at its middle portion.

The bellies of the flexor muscles of both forearms are short and rather prominent, and slightly dense to the touch, while, at the same time, they are deficient in strength, as is shown by the feebleness of the patient's grasp, and by the fact that the effort is attended by a throwing back of the hand into position of hyperextension.

There is no sign of muscular hypertrophy, except as indicated by this relatively dense consistency of the flexors of the arms, and the fact that these muscles, although short and feeble, are more rounded and prominent than would be expected.

It will be shown later that apparently hypertrophied fibres were, however, met with not only in these muscles but also in others where no outward trace of any hypertrophic process was to be seen.

The patient's skin is of a dark color, and has been for more than twenty years the seat of chronic psoriasis. The dark color is not, however, confined to the diseased portions.

The face is of the same dark tint with the rest of the body, and the muscles there are also rather feeble, although all motions are possible. In masticating food the muscles of the jaw feel weak and get fatigued. The intrinsic muscles of the hand are well preserved.

The patient, who is a very intelligent person, complains that for a long time past she has had indescribable sensa-

tions in her head, which, she says, makes it impossible for her to collect her thoughts. She also falls down occasionally, not from tripping or losing her balance, but in consequence of some cerebral cause, although she feels sure that she does not wholly lose consciousness. Ever since seven or eight years old she has found increasing difficulty in committing to memory.

The electrical examination, which was made with great care, gives the following result :—

Faradic current; facial nerves; separation of coils necessary for mimical response, 120 mm. (that is, a very weak current, the reaction being normal); ulnar nerve, separation of coils, etc., 123 mm.; media nerve, separation of coils, 105 mm.

The muscles of the thenar eminence react at 120 mm.; Extens. dig. long. at 105 mm.; flexors of forearm at 105 mm.; biceps at 105 mm. (contractions very feeble).

In other words the facial nerve, the small muscles of the thumb, and the ulnar nerve show a normal reaction. The reaction of the median nerve is apparently diminished, but probably on account of the condition of the flexor muscles. The reactions of the biceps and of the flexors of the forearm are also diminished.

The muscles of the lower extremity below the knee respond only to maximal currents, upwards of 70 mm., when the electrode is applied to the nerve, while a still stronger current than this is required to make the muscles contract by local stimulation. The reaction of the ex. quad. long. is slightly better, but the contraction is excessively slow and feeble. The reactions to the galvanic current are exactly proportionable to those of the faradic. No trace of degenerative reaction is anywhere seen.

The patient reports, with regard to her *family history*, that her father was a vigorous man, and died of cancer at the age of seventy.

Her mother was in good health, but had three paralytic attacks in late life, and died at eighty-six.

The father had two brothers. One of them began at seventeen to have similar troubles with that of the patient, and after a time became entirely crippled, so that he could not even hold his head erect, or use any part of the body, but he lived for several years in this condition, and lived to be over sixty years old. The other brother was well until about twenty-five years old. Then he had some weakness which was called rickets, and from this he never recovered. He became gradually helpless, so that he could not stand or walk, but could wheel himself around in a chair. He lived to be about sixty, and died within a day or two of the elder brother above mentioned.

The patient's paternal grandfather died from injury at sixty-five years. Health good.

The paternal grandmother died from a paralytic shock at seventy. This grandmother had a sister who became a cripple. The patient does not know at just what age her weakness came on, but she was finally so helpless that she had to be lifted in and out of bed. The family history back of this point is not known.

The patient has a brother who was in the army, and, of late, has lost his health, and is supposed to be consumptive. She has recently heard from him that he is losing the power in his legs, but cannot say exactly to what extent.

The patient had one sister who died of consumption. Four brothers have also died, but mainly from acute affections. On the mother's side the family were not considered very healthy. One branch was liable to what was called "numb palsy."

The psoriasis from which the patient herself suffered is said to have occurred in the mother's side of the house.

For the five months previous to June, 1888, the patient was treated daily with strong applications of electricity, the combined F. and G. current being used, and has also taken phosphorus, cod liver oil, and arsenic most of the time. Under this treatment there was a marked improvement, which seems to be well established.

The patient can walk very much longer distances than before, and with less fatigue. She can get her hands be-

hind her back so as to apply ointments, etc., which was at first almost impossible, and can use her hands for sewing and knitting, and feeding herself, all of which were difficult and fatiguing before the treatment was begun. Moreover, the universal psoriasis had, by June, practically disappeared, there being only a few spots on the back and on the feet remaining.

This disappearance has been gradual, having begun about February or March. The abscesses also ceased reforming early in the spring. The feeling of confusion and distress in the head also became less, especially during the last few weeks of treatment, when galvanism was applied to the head.

During the summer she was in the country, and soon discontinued the arsenic and electricity, but kept on with the phosphorous and oil.

The gain in strength maintained itself, but the distress in the head increased, and the psoriasis returned. I attribute the previous gain mainly to improvement in nutrition and nervous energy, through the tonic, and, in part, through the electrical treatment, rather than to a definite change in the muscular condition.

The electrical investigation led me, however, to think highly of the use of the combined faradic and galvanic currents, as recommended by DeWatteville, which made it possible to obtain contractions with weaker faradic currents than would otherwise have been necessary.

It was noticeable that the contractions obtained in this way were increased, not only at the moment of closure of the galvanic circuit, but during the flow of the current.

As regards the psoriasis, its disappearance may of course have been accidental, but as this had never happened before, within the patient's memory, it seems fair to attribute it to the arsenical treatment.

Two pieces of muscle were removed for examination; the first from the right biceps, at the part where the muscle was so much atrophied that a sort of excavation had taken the place of the usual bulging of the muscle; the second from the right flexor com. dig.

The biceps was one of the muscles that still responded to voluntary effort, and also to the faradic current, but slowly and feebly. The reaction to the galvanic current was also diminished, but there was no excessive action of the positive pole, nor was the contraction slower than under the faradic current.

The forearm muscle contracted better than the biceps, where the cut was made, but still slowly and feebly. The belly of the muscle was also markedly shorter than normal.

The biceps muscle was examined fresh, and also after hardening in Müller's fluid. The fresh specimens showed here and there a partial or complete loss of transverse striation, though most of the fibres had preserved their striations.

Many of the fibres were marked longitudinally by fine granules arranged in lines and spindle-shaped forms, and consisting of fat probably.

Another change also seen in both fresh and hardened specimens was a tendency to split up into fibrillæ, some of which showed transverse striations, and some no striations.

The hardened specimen was cut in both longitudinal and transverse directions, and sections stained in various ways.

The transverse sections showed, first, a very striking variation in size between individual fibres, the smallest being less than half the size of ordinary muscle fibres; the largest at least twice as large as normal fibres. The corners and edges of these fibres instead of fitting together, with fine lines between them, are rounded off and more or less widely separated, the interstices being filled up by connective tissue, containing here and there a large number of cells, and in other places but few cells.

The shape of the individual fibres also varies greatly, not only because of the direction in which they happen to be cut, but also because a portion of the fibre is often destroyed.

The muscular bundles are also widely separated by connective tissue, which often contains glistening yellowish masses, varying in size from that of a pus cell to that of a muscular fibre. When examined separately a large proportion of the muscular fibres appear to be altered.

They are marked longitudinally by fine lines, as if teased with a comb, and the fibre is thus broken up into smaller or larger fibrillæ.



There is also evidence of a gross destructive process which has sometimes gone so far that the fibre appears as if the central portion had been destroyed, leaving a cavity behind.

In other places the fibre looks as if only irregularly thinned toward its centre.

Many of the fibres have a practically normal appearance. Here and there a place is seen even in the midst of a muscular bundle, where a fibre seems to have been entirely destroyed, only its outline being left, filled with cells which are about the size of muscle cells, though varying somewhat in dimensions. The blood vessels appear, on cross sections, to be surrounded with round cells to a moderate degree.

On longitudinal sections the most striking appearance is that of the splitting up of the muscle into fibrillæ, both where the section cuts obliquely across the end of a muscular fibre, where the fibrillæ appear frayed out, as by a comb, and also in the course of the fibre, giving rise to a marked longitudinal striation, with sometimes an appearance as if the fibre had been scored with a knife.

Most of the fibres, even where this change has taken place, have preserved their transverse striation.

There is almost everywhere a great increase of the interstitial nuclei, but in some places this is greater than in others.

The sarcolemma nuclei are also here and there greatly increased in number, but not uniformly. No intra muscular nerves could be found on these sections.

At rare intervals a fibre was seen which was excessively atrophied, and presented a homogeneous, glassy appearance.

An appearance is here and there met with as of a protoplasmic mass containing several small nuclei. Apparently this is in place of a muscular fibre which has been destroyed and represents its residue.

The piece taken from the forearm was examined fresh, and also after treatment with chloride of gold and formic acid, this method being chosen in the hope of finding the terminal muscular plates.

It may be said, in brief, that a similar difference in the size and shape between the muscular fibres was found as in the fresh specimens. On the whole, however, the alterations were much less strongly marked.

A great majority of the muscular fibres had preserved their transverse striation.

It was noticeable that the hypertrophic fibres were by no means always those in which the destructive changes were most strongly marked. A small nerve fibre was found in one preparation, and traced up to the muscle, but no terminal plates were seen. The nerve fibre itself seen appeared to be perfectly healthy.

DISLOCATION OF CERVICAL VERTEBRÆ— FIVE CASES—RECOVERY WITHOUT OPERATION.¹

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THE prevailing view among earlier writers, that vertebral dislocation uncomplicated by fracture was of the rarest occurrence, if not impossible, has been materially modified during the past twenty years, and it is now generally acknowledged that this injury is by no means uncommon.

The mobility of the cervical vertebræ, together with the inclination of their articular surfaces towards the horizontal, render this region the common seat of simple dislocation, and it is probable that the list of recorded cases in this locality would be considerably lengthened were it not for the fact that many cases terminating favorably pass unrecognized through lack of familiarity on the part of the practitioner with the characteristic features, perhaps also through hesitation in making a diagnosis of so serious import. The prognosis, however, of cervical dislocation, as shown by Ashurst's tables, is not necessarily grave, particularly when the displacement is unilateral,—only eight out of twenty-nine cases of this variety there recorded having proved fatal; four having recovered without an attempt at reduction.

Since forwarding the title of this paper as it appears in the Programme,² three additional cases of cervical displacement have been brought to my notice, all terminating in

¹ Read before the American Neurological Association, at the Congress of Physicians and Surgeons, Washington, September, 1888.

² Dislocation of Cervical Vertebræ. Two cases; Spontaneous recovery.

recovery (as regards life) without operation. I shall therefore present these cases briefly before describing in detail the two which it was originally intended to report.

Of these five consecutive cases which have been treated at the Massachusetts General Hospital, two (one unilateral, the other bilateral,) are practically well, with scarcely any deformity and no paralysis; a third (bilateral) with only a moderate irregularity of the vertebral column; a fourth is out and about, though with the head bent to one side and twisted in the manner characteristic of unilateral dislocation and with paralysis causing partial disablement; the fifth with the head in a similar position, and with progressive paralysis of an upper extremity which points to pachymeningitis, on account of which the prognosis is less favorable than in the other cases.

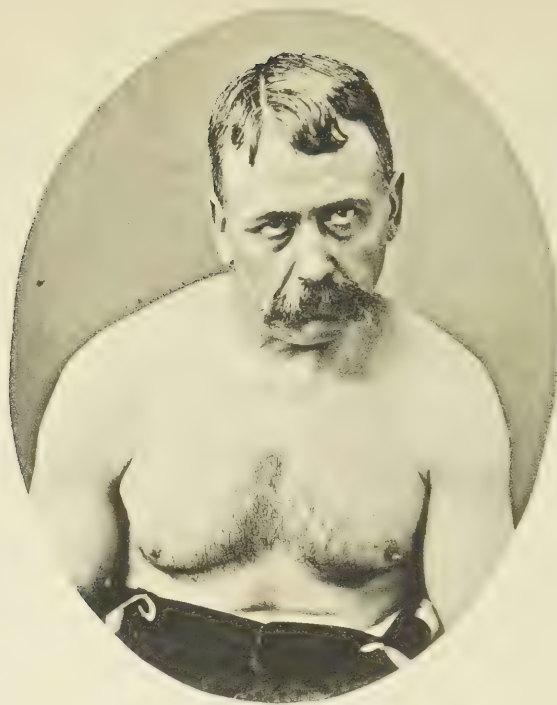
Reduction was attempted in one only of these five cases, and in this case it was unsuccessful, although spontaneous replacement occurred later.

The two cases which I shall first present briefly are of comparative rarity, in that dislocation was present with practically no paralysis.

CASE I.³—M. D., under the charge of Dr. M. H. Richardson at the Massachusetts General Hospital, six years old, is said to have fallen, twenty-five days before admission to the hospital, an unknown distance from a tree, striking on his head. He was unconscious for a short time. The head was bent forward on the breast, and deflected to the right side, the neck being held stiffly. Considerable swelling of the back of the neck followed. There was pain locally and over the occiput. There is said to have been some slight difficulty in swallowing, and some impairment in speech for two or three days after the injury, but no other symptoms. There is no other history showing a likelihood of previous cervical caries. The patient is a well-nourished child. The following notes are taken from the hospital records:—There is considerable swelling on the back of the neck, the neck is held stiffly, the head bent forward on the breast.

³ Two views in lower right hand corner of illustration.





There is a local pain and tenderness. Movement of the neck unaccompanied by the body is impossible. There is a marked bony projection, apparently of the posterior arch of the fourth cervical vertebræ. Through the mouth a projection of bone into the pharynx is felt. There is no trouble in deglutition; there are no paralytic symptoms whatever. Five days after admission he had retention of fourteen hours, relieved by a hot bath. The swelling over the neck lessened under rest in bed, and the pain disappeared. At the end of three weeks the swelling and flexion of the head disappeared, but the prominence was still felt in the neck, as well as the projection in the posterior wall of the pharynx. These were present when he was discharged, four weeks after admission into the hospital.

CASE II.—C. E., seventeen years of age. Referred by Dr. Mulligan, of Milford, to the Massachusetts General Hospital, where he was seen by Dr. J. C. Warren, at whose suggestion I examined him later at his home, when practically recovered. One year ago he fell from a trapeze about eight feet, striking the top of his head. Displacement of the neck followed, the head being bent forward on the chest and to the right; so that the chin nearly reached the shoulder. It was impossible to move the head without the body. There was pain and sensitiveness in the back of the neck. There was no trouble in deglutition, respiration, or micturition, no numbness or motor paralysis from the first. The head returned to its present position within three months. The head is now held somewhat stiffly forward, and there is an apparent shortening of the neck. Lateral motion is somewhat limited in both directions, and there is a tendency to turn the body with the head. There is a marked prominence over the fifth cervical vertebræ; the third and fourth cannot be made out. The general condition of the boy is that of perfect health, and he is able to work as before.

The following is a typical case of unilateral dislocation, followed probably by pachymeningitis giving rise to paralysis of one arm, at present increasing :—

CASE III.⁴—F. L. applied for treatment at the Nervous Out-patient Department of the Massachusetts General Hospital, in August of this year. Dr. Putnam, who recognized the nature of the case, kindly referred him to me for examination. The patient, a boy of eleven, fell about eight months ago on the ice while skating, and struck on the back of his head, which was thrown forward and turned to the left. There was no trouble in deglutition or respiration. No paralysis was noted until about three months ago, when weakness appeared in the left hand. This has gradually increased, and there is now a marked impairment in all the movements in the left arm and shoulder, the patient being unable to place his hand on his head, to grasp firmly, or to approximate the thumb and little finger. He now complains of severe pain in the back of the neck, running up into the head; there is some tenderness over the region of the fourth cervical vertebræ and above. The head is held stiffly to the left, the chin being depressed towards the left shoulder. The muscles are quite tense on the right and comparatively lax on the left. Rotation and flexion of the head are possible only in a very slight degree. There is a prominence over the fourth cervical vertebræ; a prominence, which appears to be the spinous process of the second, is found to the right of the median line. There is atrophy of the scapular muscles, most marked on the left.

In marked contrast to these cases, as regards paralytic symptoms, are two cases which formed the original subject of this paper, and which I shall present more in detail.

The first of these cases was one of typical bilateral dislocation, the third cervical vertebræ being dislocated forward on the fourth, paralysis of all extremities following, as is commonly the case, through pressure of the posterior arch on the spinal cord. The interesting feature is the fact that spontaneous replacement and recovery ensued after failure of operative interference, and after progressive paralysis and enfeeblement lasting over a period of fifteen months.

⁴ Two views at left of illustration.

CASE IV.—A. W., cook, single, thirty-five years of age was admitted to the Massachusetts General Hospital in the service of Dr. J. C. Warren, with whom I saw him from time to time during his stay. He was seen also by a number of other physicians, including Dr. J. J. Putnam and Dr. M. H. Richardson.

The history was as follows:—On January 3, 1885, he fell down a flight of steps backwards, striking his neck on the edge of a doorpost. His head was thrown forward with the chin elevated, in which position it remained up to the time of entrance. He lost consciousness for six hours, and remained in bed about one month, complaining principally, apart from the displacement and rigidity of the head, of general weakness, numbness, and stiffness of the legs when getting up, and a slight twitching in the hands. There was no trouble in breathing from the first. He entered the hospital March 30th, about two months after the accident. He complained at that time of pain in the shoulders and across the back, and of gradually increasing weakness. The head was projected forward with the chin elevated. There was a marked prominence over the fourth cervical vertebra; above this point the spinous processes of the vertebræ were less prominent than normal. Digital examination of the throat showed a projection in the posterior pharynx. The patient was able to walk, but with a spastic gait. Ankle clonus was present; the patellar reflex was so greatly exaggerated that tapping the tendon produced a continuous clonus. Respiration was normal. There was at that time no objective disturbance of sensation. The grasp was weak on both sides, as well as extension of the wrist. The supinator longus was strong, as were the muscles of the upper arm, excepting the triceps on the left, which were feeble. There was no marked reflex in the arm. Every attempt at movement of the legs caused tremor. Flexion and extension of the thigh was fair on both sides; the tibialis anticus and gastrocnemius were fairly strong; the peroneal muscles weak. The legs were rigid; there was no atrophy or coldness. The plantar, abdominal, and cremaster reflexes were normal. The

pupils were equal and reacted to light; there was nothing abnormal about the face. The respiration was 20, the pulse 86, the temperature normal.

Three days after, operation was undertaken by Dr. Warren. The patient was etherized; the cervical vertebræ were extended by pulling the head in one direction and the body in the opposite. No distinct snap was felt, but the prominence of the vertebræ was considerably diminished. The neck was held in position by bandaging the head and body to a broad leather splint. The second day after the operation there appeared to be an improvement in the patient's condition; the grasp was stronger and the ankle clonus less marked. On the fourth day the apparatus was omitted. On the sixth day careful examination by myself showed no improvement over his previous condition. On the sixteenth day the patient was gradually losing ground; he was growing feebler, and the cervical prominence, together with the peculiar manner of holding the head, had returned. Sensation was impaired in legs and arms. One month later the condition was not changed, excepting in the direction of increased feebleness. After two months the patient could not stand on his feet without assistance. After two and one-half months the head of the bed was elevated, and extension was applied to the neck with halter and weights. This apparatus was removed five days later. At the end of three months the patient was gradually failing. Bladder symptoms had appeared in the form of retention. There was tonic spasm of the legs. Sensation in the legs was lost to the groin, and in the arms to the middle of the upper arm. Four months after operation the patient was completely helpless and unable to grasp anything firmly in the hand; he was much troubled by constipation, and suffered at intervals from retention, which required the use of the catheter. At the end of four months and one-half further surgical interference was considered inadvisable, and he was discharged from the hospital, but was allowed to remain at the Convalescents' Home until ten months after the accident, when he was taken to the Almshouse, where he remained without im-

provement for three months longer. At the end of this time, that is, about fifteen months after the accident, while taking a lukewarm bath, ice-cold water being meantime thrown upon his back by a syringe, he suddenly felt a sensation like an electric shock. He was rubbed down and put to bed, and the galvanic current was applied to all extremities.

The next morning he found he could rise in bed. Cold water was thrown after this in large quantities on his back every other day, after which the galvanic battery was applied. Improvement was steady and rapid, and within a month he was at work in a restaurant, where he was seen by Dr. Richardson. The head had become gradually replaced during this time. No further symptoms appeared.

I examined the patient carefully three years after the accident. The gait was normal and free, the tendon reflex was normal; there was no rigidity or ankle clonus; all movements of the extremities were perfect, and the lightest touch was everywhere felt. The head was held rather stiffly, canted slightly to the left, with the chin elevated and turned to the right, the whole head being held somewhat forward. The fourth and fifth cervical vertebræ were rather prominent, though no more than is sometimes found in health. The spinous processes above were distinctly felt; there was no prominence in the posterior pharynx.

The duration of life and final recovery in this case are quite remarkable. Liddell,⁵ under the subject of bilateral dislocation of the last five cervical vertebræ, in discussing the question of operation, says: "In pursuing such a course (operation) I would be guided by the following considerations: (1) *The almost complete certainty of a fatal termination within two or three days if an expectant plan of treatment be followed.* Of thirty-six perfectly analogous cases treated at Guy's Hospital, all died within seventy-two hours. . . ."

That respiration was unaffected is not unique, a fact probably due to the departure of the roots of the fourth cervical nerve from the cord above the articulation of the third

⁵ International Encyclopædia of Surgery, Ashurst, 1884, vol. 4.

and fourth vertebræ. This is illustrated by the case of Cushing, quoted by Shaw,⁶ in which a patient lived two days after dislocation of the third or the fourth cervical vertebra. The rule is, however, as stated by the same author (Shaw), that where the cord is crushed above the level of the fourth cervical vertebra, that is, above the origin of the phrenic nerve, it may be considered certain that instant death will ensue.

The last case is one of unilateral dislocation of atlas on the axis, produced by violent muscular exertion in wrestling. I saw him for the first time, one year after the accident, when he was sent to the neurological department of the Massachusetts General Hospital for an opinion, by Dr. H. W. Boutwell, of Manchester, N. H. Dr. Warren saw him with me in consultation at the hospital, and considered it an undoubted case of dislocation. I afterwards saw him in Manchester with Drs. Boutwell and Wilkins.

CASE V.⁷—L. C., forty-four years of age, a weaver. The patient is a stout man of short stature, but unusual muscular [development. While wrestling, about a year ago, he put up his right hand to throw off his opponent's arm, which was about his neck, his own head being bent at the time to the right. He made a violent effort, and as he did so suddenly felt dizzy and heard something snap, as did, also, the bystanders; he immediately found that his head was fixed on one side (to the right). He did not lose consciousness. When given a glass of water shortly after, he found he could not swallow. His articulation was indistinct, he "talked thick," but had no difficulty in choosing words; he does not think he talked through his nose, but as his speech is still decidedly nasal, without his realizing the fact, this condition probably obtained at the time of the accident. About an hour and a half afterwards, on trying to eat, he found that food caught between the teeth and the cheek on the right, and that when caught he could not remove it with the tongue; liquids ran out at the nose. The physician who was summoned about an hour and a half after first no-

⁶ Holmes's System of Surgery, 1881, p. 807.

⁷ Two views in upper right hand corner of illustration.

ticed that the tongue was deviated to the right. There was trouble in breathing from the first. The patient was unable to whistle, which he could do perfectly before. There was constant tinnitus auris dextræ for some time. There was a large protrusion over the triceps which gradually subsided (rupture ?), but a weakness of the right arm has persisted.

Physical examination.—The patient is a stout man of excellent muscular development. The gait is normal. There is no paralysis of the lower extremities, motor or sensory ; the skin is everywhere natural, and the flesh well nourished ; there is no rigidity ; the tendon reflex is active, but not exaggerated ; there is no ankle clonus. The grasp of the right hand is not quite as strong as that of the left, and the same is true in a slight degree of all the movements of the right arm, though there is no localized paralysis, wasting, or coldness, and no disturbance of electrical reactions. The head is held stiffly bent to the right, with the chin turning somewhat to the left, the face looking downwards, the position being somewhat similar to that produced by contracture of the right sterno-cleido mastoid muscle. This muscle is, however, quite lax, as are all the muscles of the neck on the right side, while those on the left are comparatively tense ; this is quite apparent in the photograph. The head, besides being bent to the right, is set off, as a whole, to the left, as is also apparent in the photograph, especially the posterior view. Attempts to straighten the head forcibly cause pain under the occiput on the right side, a point which seems somewhat sensitive to pressure. A certain amount of rotation of the head is possible, principally to the right. The extreme excursion of the end of the nose is, however, only three and one-quarter inches. Flexion and extension of the head are practically impossible. No weakness is detected in the sterno-cleido mastoid or trapezius ; no prominence is felt in the fauces, nor depression over the spinous processes of the vertabræ. The tongue is deviated markedly to the right and cannot be moved to the left ; it is also greatly atrophied on the right side, comparatively few fibres being left. The electrical reaction on the left side of the tongue is normal ; on the right there is only very slight re-

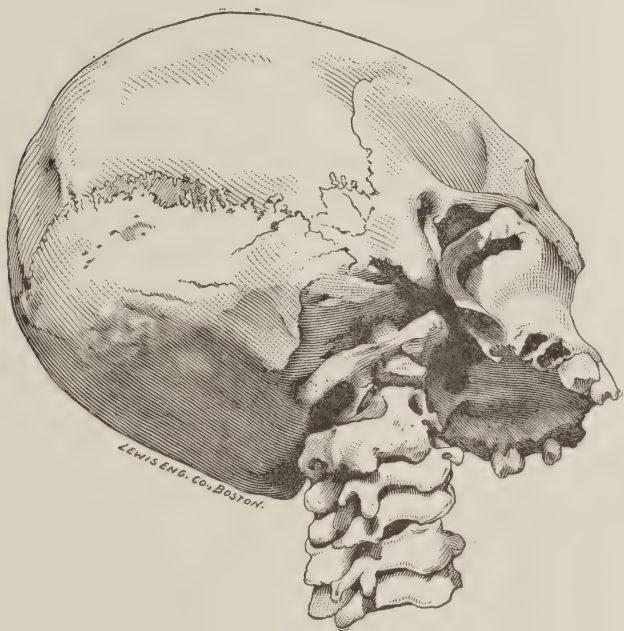
action to the faradic current, the response being apparently that of a few intact fibres. The uvula is deviated to the left and the right side of the palate reacts only slightly to mechanical stimulus. The speech is thick and somewhat nasal in character. The patient can now whistle, though not perfectly; there is still some difficulty in mastication, the food lodging in the cheek on the right. The forehead wrinkles normally and the eyes shut well, there being only a trace of paresis remaining in the facial muscles, all of which react perfectly to the faradic current. Movements of the eyes are perfect. The right pupil is slightly larger than the left, and reacts less promptly to light. Vision is good in both eyes; the fundus is normal. There is no loss of sensation in the face or elsewhere. The watch is heard at a distance of four inches on the right, of ten inches on the left. Both drumheads are retracted, the reflex dull on the left and broken on the right; bone conduction is normal. Examination of the vocal cords is impossible on account of the elevation and immobility of the back of the tongue. There is no special rigidity or exaggerated reflex condition of the right arm, but the muscles are not quite as firm as those on the left; the right forearm measures, however, the same as on the left, and the upper arm a quarter of an inch more.

The lesion in this case was probably a unilateral dislocation of the atlas on the axis, which "consists essentially" (Liddell)^s "in the displacement of the inferior articular process on one side of the cervical vertabræ from the corresponding superior articular process of the vertebræ which lies next below; this causes the victim's face to be turned towards the side opposite to that on which the luxation is situated."

The diagnostic features correspond closely with those described by the same author, and consist in the twisting of the neck, the fixed position of the face, with the chin pointing towards the left shoulder (the luxation being on the right), the immobility, and the tenseness of the muscles on

^s Ibid., p. 732.

one side of the neck, combined with relaxation of those on the other side. Paralysis of an arm, and sometimes of the lower extremities, may also occur in greater or less severity; in our case the paralysis of the right arm alone being observed from the first, and this slight in degree. I have found no record of paralysis of cranial nerves like that from which this patient has suffered; the cause being probably, in this case, an extensive effusion in the region of the medulla, pressing on the hypoglossal, glossopharyngeal, facial and auditory nerve roots.



The position of the head resembles very nearly that of the skull in the accompanying photograph, which I was able, through the kindness of the curator, Dr. Whitney, to obtain from a specimen in the Warren Anatomical Museum, at the Harvard Medical School, which was called to my attention by Dr. J. C. Warren. The description in the catalogue is as follows:

"970. *Vertebræ*.—Fracture of the second left articulating process of the *vertebræ*, with dislocation of the atlas (co-ossified to the skull) downwards and to the left. The two *vertebræ* firmly united in the new position, and the head tipped to the left at a considerable angle. A young adult. One of the incisors had not completely come down, although the skull is evidently of an adult—J. Mason Warren Collection."

Unfortunately the ante-mortem history of the case is not known, the skull having been purchased in Paris by Dr. J. Mason Warren. The intact odontoid process is plainly seen in the posterior view, and it is evident that, notwithstanding the extreme lateral displacement, a canal was left sufficient for the passage of the spinal cord. That such a canal was left in our case is evident from the absence of paralysis of the extremities, excepting in a slight degree in the right arm, and from the fact that respiration was unimpaired. The transverse ligament cannot have been ruptured, as the escape of the odontoid process causes instant death by pressure on the cord, the unfortunate victim being practically pithed. Our case differs from that of the anatomical specimen in that there was probably no fracture, but a simple unilateral dislocation, and also in that a false joint has been formed, allowing some lateral rotation, whereas in the anatomical specimen the bones are firmly fixed in their new position. Quite extensive hemorrhage probably accompanied the dislocation in our case, as it can hardly be supposed that the paralysis of cranial nerves resulted from direct pressure. The difficulty in swallowing resulted, perhaps, in part from mechanical obstruction in the pharynx, although it is probable that the glosso-pharyngeal nerve was involved in the hemorrhage; indeed it is not improbable that the paralysis of the palate results from injury to this nerve, which, it has been suggested, may furnish the fibres (generally credited to the facial) which pass through the Vidian, and supply the levator palati and azygos uvulæ. That the hypoglossal and facial nerves were included there can be no doubt, as the paralysees resulting have in great

part remained permanent. It is notable that the auditory nerve suffered so little, the only evidence of its implication being the tinnitus aurium and temporary increase of deafness in the right. The deafness now existing in the right ear, like that in the left, is that which would be expected from chronic catarrh, the bone conduction being equally good on both sides, with a tendency in the direction of increase.

Unfortunately, apparatus has not been at hand during the examinations I have been able to make, to test for various tones, or for high tones, or possibly some defective fibres might have been detected.

The recovery in this case, as in the other unilateral cases, is less remarkable than that of the patient with bilateral dislocation, the prognosis in these cases being, as already stated, much more favorable on account of the spinal cord being less liable to damage.

Perhaps the most interesting feature in the case is its ætiology, the manner in which the dislocation was produced being probably unique. The causes mentioned in the tables above referred to consisted in turning the head quickly, falling on the head, falling on the neck, a bundle slipped on the shoulder, a fall in running, direct violence, being thrown against a wall, tumbling heels over head on a bed.

It is not improbable that an attempt at early reduction would have produced a more favorable result, but in consideration of the absence of imminent symptoms, and considering also the comparatively favorable course of cases of unilateral dislocation without operation, the patient is perhaps quite as well off as if operation had been attempted. Whether the nature of the trouble was recognized at the time of the accident I do not know.

CEREBRAL SYPHILIS.¹

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NO class of cases presents such variety of symptoms as exist in syphilis involving the cerebral structure.

Rumpf divides syphilitic diseases of the brain into three classes : first, those involving the cranial bones ; second, new growths involving the brain or its membranes, and, third, specific disease of the cerebral vessels.

In my paper this evening I do not intend to take up the subject of syphilitic tumors or inflammation of the membranes, but shall confine my attention more particularly to disease of the vessels.

Many of the earlier appearances of syphilitic disease of the nervous system, as during the secondary stage, are probably due to simple hypertrophy of the tissues affected.

Virchow describes two conditions as existing pathologically, the one a simple hyperplasia, and the other a heteroplasia ; the latter only, presenting the characteristics of a new growth, distinct and foreign to the part affected, so that for instance, a syphilitic node affecting the cranial bones is of the same distinctive character as a gumma, involving the dura or the pia.

This new growth is the same in the tertiary stage, following, perhaps, thirty years after the original infection, as in the secondary stage, leading one to believe with Rumpf, as opposed to Hutchinson and Ricord, that the virus itself is still active, and that these changes are not due to the effect of a poison which has expended itself, but left its impress on the system.

¹ Read before section on Practice, of the N. Y. Academy of Medicine, Dec. 18, 1888.

This view is also clinically sustained, as we find many cases of hereditary syphilis transmitted late in the course of the disease, passing through precisely the same class of symptoms as when it is transmitted in the early stages.

The pathological changes in the nervous system in hereditary syphilis do not differ from the acquired form, except that in the former gumma are rare, while in the latter it is the most frequent form of tumor cerebri.

Hutchinson says that cerebral syphilis is rare in the hereditary form, but Dowse and others disagree with him, and find material changes very frequent.

It was long thought that the cranial bones were always the original seat of the cerebral lesion, and although it is frequently so, yet we now know that the dura or pia, or the brain itself may be independently affected.

Meyer considers matting together of the dura and pia with softening of the cortex as almost pathognomonic of syphilis, as in pachy-meningitis, from other causes this rarely occurs.

Characteristic of the pathological changes in specific endarteritis, as distinguished from atheroma according to Heubner, is the fact that in atheroma the large vessels only are affected, and long tracts of the arteries are involved. Occlusion occurs by thrombosis, never by narrowing of the lumen of the vessel; there is often, at first, even a widening of the lumen of the artery. The process is very slow and consists in simple hypertrophy resulting in calcification, there being no new growth.

In syphilis we find both large and small arteries affected, with a tendency to disease of the small and middle sized vessels. We may indeed have thrombosis, but it is secondary to the narrowing and final occlusion of the vessels from extension of the new growth inwards. Rarely, however, is the endothelial lining of the vessels involved.

The action of the syphilitic virus seems to cause a great increase of cell growth through irritation of the endothelium and commences in the inner coat or intima. When a new growth surrounds the vessels, as in syphiloma or meningitis of the base, the adventitia may be primarily affected.

Arterial disease may occur entirely independent of syphiloma, and is rapid in its course, differing thus from atheroma. According to Dowse the vessels of the pia mater are very often thus affected, leading to softening and atrophy of the cortex and causing thereby a class of symptoms differing from those in which the dura is involved.

The tendency to the formation of new vessels with thin walls so characteristic of all granulation tissue is also present here, so that we have at first a tissue richly supplied with vessels. This of course depends somewhat on the seat of the growth, as in the dura, or between the cranial bones and the dura the tissue is compact, and but feebly supplied with blood, while in the arachnoid and especially at the base the vessels are very numerous.

This rapid cell growth, however, leads to pressure on its vessels, and interference through anæmia with its further extension, so that it falls into caseous degeneration, never into suppuration.

This, in a degree, is the same when the disease is limited to the arteries, further extension may be prevented and even retrograde changes may take place, leading to restoration of the lumen of the vessel and disappearance of symptoms. This might readily occur when the cortex is involved, the collateral circulation preventing softening, when, however, the terminal arteries passing to the ganglia and internal capsule are affected, the result may be permanent.

Rumpf places the origin of the endarteritis in the middle coat of the arteries, and insists on its character of granulation tissue similar to that found in all syphilitic growths.

This cell growth may extend inwards, causing occlusion of the vessel and thrombosis. He admits the difficulty of differentiation microscopically from atheroma, but believes that a special bacillus can here be found which is the same for the primary chancre and other later growths.

He shows a drawing of syphilitic disease of the vessels, in which the narrowing of the vessels is clearly delineated, corresponding very closely, microscopically, to a section of the basilar artery of a case whose history I will read later.

In the discussion on a paper by Dr. Anderson before the

British Association, on syphilis of the nervous system, Dr. Ross gave the following order of selection in cerebral syphilis :

The cortical area of the middle cerebral artery giving rise to Jacksonian epilepsy ; the cortical area of the anterior cerebral in which somnolence was present and epileptic seizures infrequent; the subarachnoid space at the base, in which some affection of the cranial nerves ushered in the attack, the disease later extending to the vessels passing into the anterior perforated space, resulting in occlusion of the vessels of the ganglia softening and hemiplegia ; the subarachnoid space at the posterior border of the pons, in which case the disease is ushered in by paralysis of the 6th, 7th and 8th cranial nerves, and a tendency to fall toward the side of the lesion.

In giving a description of the symptoms of cerebral syphilis I shall refer to those of a general rather than a focal character, not taking up tumors as they must be similar in their direct effect to those of any other growth.

I would speak first of the psychical changes present.

These may be sudden in their onset, but more often gradual, extending over months, or even years. We notice a general loss of mental power, the ethical nature seems to suffer, the memory weakens, there is often a feeling of loss of power of performance resembling that of neurasthenia cerebialis; the nature seems changed, there is often developed a tendency to brutality and selfishness, and many acts of intemperance and immorality are committed entirely foreign to the known character of the patient.

The loss of memory, of the association of ideas, and therefore of the judgment, is probably explained pathologically by Clouston's statement that the cortex cells and the associating fibres undergo degenerative changes, such as we know take place in general paresis.

Insomnia, independent of cephalalgia, is one of the early symptoms and always a serious one, or again somnolence or even stupor may occur during the daytime, periodically or continuously.

This is not to be confounded with the coma of an apoplectic seizure.

Cephalalgia is perhaps one of the most frequent of all the early symptoms and indicates an affection of the cranial bones or of the dura mater, it may be worse at night, and is always accompanied by a feeling of mental weakness and confusion. Cephalalgia is not always present, indeed is usually absent when the lesion is in the arteries or in the brain itself; the psychical confusion, however, always exists.

The cranial nerves are frequently affected, especially the 3d, 6th, 5th, and the optic. We may have these nerves bilaterally or unilaterally affected, thus ptosis of one side alone, or again the 5th nerve of one side only involved.

Bulbar paralysis, according to Buzzard, may occur, although I believe it is rare. Hutchinson says the 7th alone is rarely affected, and that he has never seen a case, although others (Jackson) have reported them. He is equally strenuous on the diagnostic value of an affection of the 5th as indicating syphilis.

In tumors or meningitis of the vertex these nerves necessarily escape, while where the base is involved many, including the facial, are affected. It is however in disease of the vessels unassociated with tumors or meningitis, we often find the single paralysis of the cranial nerves.

Epileptic seizures are often present, and, as the inflammation is usually diffuse rather than circumscribed, they are more often general than focal. This is seen in cases of lepto-meningitis, with escape of the dura and cranial bones. Following these attacks the coma is apt to be profound, with often a maniacal condition and paralysis transient in character.

In regard to another of the early symptoms, I would direct especial attention to the eyes.

In the secondary stage of syphilis, as we know, the iris and cornea are frequently involved, and usually symmetrically, and in doubtful cases it is well to bear this in mind.

I would especially refer to choroiditis and retinitis as indicative of an already existing affection of the cerebral

vessels. A cloudiness of the disc, appearances of atrophic spots in the fundus, traces of a post retinitis or choroiditis, gummatous enlargements involving the terminal vessels, aid in a positive diagnosis.

F. Ostwalt, in an excellent paper in the *Berl. Kl. Wochenschrift*, No. 45, 1888, dwells particularly on these points, and draws a very important conclusion from them, that they are secondary to changes in the carotid artery, the ophthalmic artery giving symptoms much earlier than the cerebral vessel with its more complete collateral circulation.

As a diagnostic point, an interference with vision in any part of the field, or a general loss of acuity of vision in the young, should lead to the suspicion of cerebral syphilis. The writer relates a case where two or three such attacks preceded by several years a fatal hemiplegia.

Hutchinson notes that in hereditary syphilis, often as late as the tenth to the sixteenth year, corneal affections, usually symmetrical, may occur; and that often, when no signs of the disease have shown themselves during the first two years, later, mental weakness may develop, or melancholia, mania, or dementia. He also mentions a symptom which I have not found generally referred to—the appearance at this age of deafness without local cause, for which there seems to be no cure.

The early recognition of symptoms is of great importance, as later treatment cannot be of much avail.

Transitory paralysis and paralysis of parts widely separated are strongly diagnostic of specific disease.

An hemiplegia coming on without loss of consciousness, in which the patient can relate the commencing numbness, and tingling, and final paralysis as it spreads from the arm to the leg, especially if the condition has been preceded by irritability, loss of memory, cephalalgia, confusion, somnolence, in a person under forty, is probably specific.

Many, if not precisely this class of symptoms occur in the aged, due to thrombosis.

It is not unusual in these cases to observe a tendency to somnolence or even stupor, passing into coma and paraly-

sis, the latter often being only temporary. In these cases, however, we have the advanced age, the atheromatous arteries, or some chronic kidney disease pointing to the cause.

In examining some fifteen or twenty cases of hemiplegia from various causes, I found, out of six of specific origin, five affecting the left side; and while this could not be taken in any sense as the proportion, yet it suggests one fact, that hemiplegia in syphilis is more often on the left side than in other cases; in other words, it may as often affect the vessels of one of the hemispheres as of the other.

In the discussion on Dr. Anderson's paper, before alluded to, Clouston gives several principal forms of brain syphilis. First, where we have a short delirium during the secondary stage; second, acute delirious mania during rapid growth of a syphiloma in the cortex, in the region of the vertex anteriorly, with rapid progressing softening, the patient dying within three weeks of the incidence of the disease; and third, the most common form he ascribes to syphilitic endarteritis, the mental symptoms being immorality, sudden impulsiveness, insane suspicions, and gradual dementia accompanied by convulsions and paralysis.

Buzzard says that multiple symptoms are diagnostic of specific disease of the nervous system, the occurrence of a lesion involving two parts of the cerebro-spinal system being rare except in syphilis.

Transitory paralysis may suggest hysteria, as in a case reported by Heubner, in which extensive specific affection of the arteries and membranes was found post-mortem.

Cerebral syphilis may also be confounded with neurasthenia cerebralis, the mental inactivity, depression, and apathy rendering it very difficult of diagnosis, especially in those cases where headache is absent.

It is especially in these cases that a careful inquiry into the previous history becomes necessary, and an examination of the field of vision. The presence of signs of a previous iritis or choroiditis, also of any signs of thickening or of nodes affecting the skull, clavicle, sternum, or tibia, of

old scars, especially if pigmented or concentric, of atrophy or hypertrophy of the testicle, of lesions of the tongue, etc., is indicative of specific disease.

Epileptic seizures after twenty years of age should lead to the suspicion of syphilitic origin when injury or other causes can be excluded. As in idiopathic epilepsy, the cases are most common between ten and twenty years.

According to Hughlings Jackson, in these cases we may have only a slight contraction of the fingers or the face without loss of consciousness, or, again, very rapid succession of fits with intervals of deep coma.

Rumpf lays great stress on the pupil changes as a diagnostic point in cases where no focal lesion is present, an inequality of the pupils, or the more active response to light of one than the other.

The Argyl-Robertson pupil, one of the earliest symptoms in tabes, he considers also an initial symptom in cerebral syphilis, and quotes Moeli and Oppenheim as agreeing with him. This is of especial importance in a differential diagnosis from neurasthenia cereбрalis, and the writer relates a case which recovered under specific treatment, in which this was the only symptom pointing to a syphilitic origin.

I consider it of great value, and have tested it in several cases which subsequent treatment and examination proved to be specific.

In conclusion I would detail briefly a few cases that have come under my care in private and hospital practice which more or less illustrate many of the symptoms and pathological changes given above.

CASE I.—A. M., colored, female, æt. 35; unable to obtain family history. Patient married at 16; sterile; specific history. Her sister says that the patient was a temperate and hard-working woman up to the summer of 1887, when she became changed in character, and frequently came to this city from her home in Brooklyn, and became intoxicated and went with lewd company. Patient had never complained of headache or trouble with sight or hearing, so far as she knew. Patient was admitted to the workhouse in

September, 1887, for disorderly conduct. The house physician reports that she was irritable, indecent in speech, confused, and did not seem to hear or understand what was said to her. Soon after admission had an epileptic seizure, general in character, followed by deep coma and mania in which restraint was necessary.

Following attack patient was blind, groping about and knocking into objects in her way. Her condition remained the same until January, 1888, similar attacks occurring every three weeks. I saw patient the day following the last seizure—condition partly comatose; position in bed one of flexion; she could be roused, but would obstinately cover herself with the bed clothing, and laugh in a silly manner; reflexes normal, and as far as could be ascertained no loss of response to touch, pain or temperature tests. There was a slight persistent tremor of right side, but no paralysis. Patient died two days later in deep coma, following a maniacal attack.

Post-mortem examination showed the following :

Skull normal, no thickenings or exostoses; dura mater not adherent or thickened; slight leptomeningitis over convexity of both hemispheres parallel to longitudinal sinus. Extensive softening and atrophy involving bilaterally and symmetrically the parietal lobes posteriorly to the motor area; the left occipital lobe much atrophied.

The vessels supplying these parts, *i. e.*, the branches of the middle cerebral and posterior cerebral were occluded or narrowed and the walls thickened.

Microscopical sections of the basilar and middle cerebral made by Dr. J. S. Thacher, showed thickening of the walls of the vessels, the intima and middle being principally involved and the lumen encroached upon.

His report is as follows :

"The intima is in spots very markedly and irregularly thickened, appearing to consist in such places of new connective tissue containing some blood filled spaces.

"Some of the deeper portions of such thickened patches showed clusters of large pale staining, coarsely granular cells which I took to be fatty degenerated cells, others of the deeper portions showed masses in which no structure could be made out.

"The media was in circumscribed places much thinned, making a kind of hole filled by thickened intima.

"Beyond this I detected no change in the media and none in the adventitia."

I present these sections along with another from a case of known specific history, in which the extension of the growth into the lumen of the vessel is very beautifully seen.

CASE II.—A. B., merchant, æt. 42. Patient enjoyed good health up to two years before coming to me. Patient complained of cephalalgia diffuse in character, vertigo, depression, incapability of sustained mental work, and insomnia, in short, many of the symptoms of so-called neurasthenia.

Patient denied syphilis, and with the exception of some tenderness on percussion over the vertex I could detect no signs of it. Under moderate doses of potass iodide complete recovery.

CASE III.—C. D., female, æt. 42. Patient had complained of trigeminal neuralgia for weeks, and had received the usual remedies. On examination I found the pupils unequal and slow in response to light, although responsive to accommodation ; patient also mentally dull.

Suspecting syphilis large doses of the iodide were ordered with complete recovery. A more careful investigation into the previous history also sustained the diagnosis.

CASE IV.—E. D., female, æt. 29. Saw patient for first time while affected with chorea and iritis of both eyes. Syphilis was denied, but I found on examination enlarged glands on the neck and several scars over the body. Patient excessively irritable, refusing food or medicine, remaining in bed in the daytime only to disturb the ward at

night with her hallucinations. Later knowledge showed that she had been in the insane asylum for one and a half years.

Under potass iodide and calomel—small doses—chorea and iritis much improved.

In regard to treatment the iodide must be relied upon. Large doses, up to 300 or 400 grains daily if necessary. Often when this fails, mercury, in conjunction with it, either by inunction or the hypodermic method, seems to be very effective. In all these cases the nourishment should be carefully watched and carried to as high a point as possible.

TUMOR OF THE CEREBELLUM.¹

By J. A. BOOTH,

ASST. PHYSICIAN MANHATTAN EYE AND EAR HOSPITAL AND VANDERBILT CLINIC.

THE subject of intracranial growths is of interest to us all; and no one appreciates that fact more than he who is fortunate enough to have such a case under observation. Knowing the importance of faithfully recording and reporting each and every case, especially when an autopsy has been obtained, I take pleasure in presenting the following history.

Sarah D—, aged ten years. Seen in consultation with Doctor Z. S. Webb on February 19, 1888.

Parents healthy; no family history of cancer or tuberculous disease. The child had not had any previous serious illness. When three years old, she fell down a flight of steps, striking her head upon the stone walk. The fall could not have been a serious one, as the parents did not recall the accident until after her death, though questioned carefully before as to traumatism.

About November 1, 1887, the parents noticed that she was losing flesh and becoming very pale.

Ten days later she came in one afternoon, complaining of chilliness and headache, followed by vomiting without any nausea. She slept well that night and seemed perfectly well the next day. On November 15th and 20th she had similar attacks of headache and vomiting. Each attack seemed more severe, and the recovery not so complete. She was also becoming very irritable—did not wish to play, talk or be disturbed. A little later the father thought she carried her head as though the neck was a little stiff.

From January 15, 1888, the child commenced complaining of pain in the back part of head and neck, principally in

¹ Read before the Neurological Section of Academy Medicine, Feb. 8th.

the morning before getting up, but always wanted to be dressed and go down stairs to her meals. She now spent most of the time in her mother's lap. The parents had not noticed any staggering in her gait, nor did she at any time complain of dizziness.

Examination (February 19, 1888).—Child dressed and sitting in mother's lap; head bound with cold compresses. She is dull, listless, complains of severe head pain, and does not like to be disturbed; is too weak and miserable to get down and walk. Speech normal; tongue straight; no tremors; vision seems normal to finger test; pupils dilated; no reaction to light. There is a slight paresis of right external rectus; no nystagmus; no changes in the fundus. Grasp of hands fair; no paresis of face or limbs; knee-jerks absent; no anæsthesia. There is marked sensitiveness to touch all over head, and especially on back of neck, just below occiput. The posterior cervical glands are quite large. Temperature in axilla $101\frac{3}{8}$. Pulse 100, irregular.

Diagnosis.—"Tubercular meningitis."

Tumor of the cerebellum was considered; but the absence of optic nerve changes and the presence of an elevated temperature with an irregular pulse, led me to give the former opinion. Besides many of the facts in the previous history of the case I was not able to obtain until later, especially as to the vomiting.

Patient was ordered ten grains of the iodide of potassium in milk every four hours, this dose to be increased five grains each day.

February 24th.—General conditions worse. Pain in head still present; vision same; pupils not so large and react to light. Paresis of ext. rectus has disappeared. Temperature 100; pulse 120; respiration 18. Ophthalmoscope showed beginning optic neuritis; vessels small and indistinct. Is taking to-day thirty-five grains of potassium iodide every three hours in milk. Vomited after second dose. To take grains twenty as before.

March 5th.—First well marked convulsion occurred, which was followed by many others, five or six during the

day. These consisted of tonic spasm of limbs, drawing up of right side of lip and also of nose; left eye wide open and right eye tightly closed. Low moans and sometimes a loud scream would accompany these attacks. Father states that on two occasions only was consciousness entirely lost.

The symptoms certainly point to an intracranial growth, and are probably due to a cerebellar lesion.

March 9th.—Very much prostrated; vomits the iodide, which had been reduced to ten grains every three hours. Temperatrre 100; pulse 118, thready and irregular. Stimulants given as necessary, and patient ordered syr. ferri iodide, twenty drops three times a day; also inunctions of cod liver oil.

March 10th to 20th.—There is a slight improvement. Vomiting ceased; appetite is good; patient swallows her food easily. Convulsive seizures less frequent and less severe. She passes her urine involuntarily and the act is generally accompanied by a seizure as before described.

Examination of Urine—1018, alkaline. Traces of albumen; no sugar or casts. The abdomen, which had been sunken in, rounded out; and the whole body seemed to flesh up once more. She is still very irritable; repeats the words of others and any sounds she hears outside; also uses strong language, quite often saying Devil! and even worse. The use of these expressions quite surprise and shock the parents, who cannot imagine where the child could have heard them.

March 25th.—The only new symptom is dimness of vision; does not distinguish objects beyond four feet. Ophthalmoscopic examination shows advanced atrophy of both optic nerves.

July 1st.—Child is entirely blind; lies most of the time in a semi-comatose state; occasionally attacks of petit-mal. There is now paralysis of right side of face, partial paraplegia and paresis of left arm. The following bulbar symptoms have also appeared: Dullness, impaired articulation, difficult deglutition, and polyuria.

August 1st.—Emaciated to an extreme degree; cannot swallow; is fed with a tube. Left arm and both legs con-

tracted and rigid. There has been a gradual enlargement of head, and there is now some separation of coronal and sagittal sutures. Slight exophthalmus present. Patient died on August 17, 1888.

Autopsy on August 18th, nine hours after death, the head only being examined.

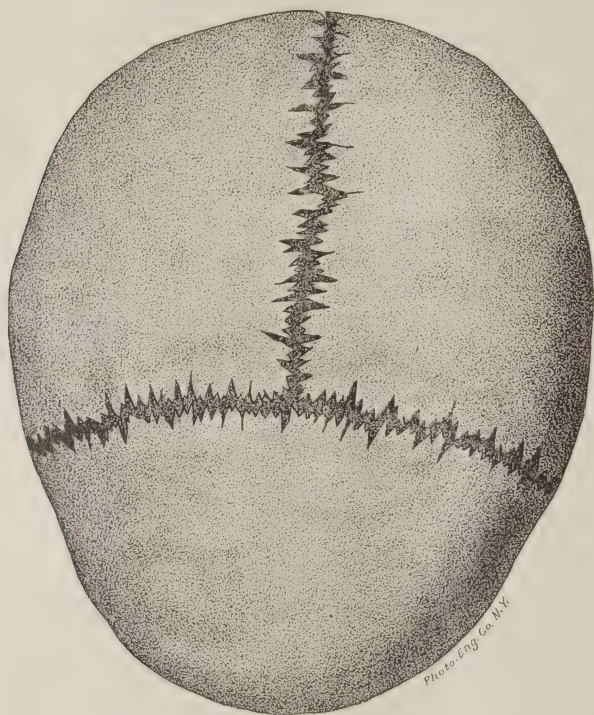


FIG. I.—Degree of separation of the sutures.

The whole head was very much enlarged, the frontal and parietal bones being very thin and separated at the sutures to a marked degree (Fig. 1). The dura mater was very thick and distended. On puncturing this, a large amount of clear fluid gushed out. The convolutions were flattened and whole brain was pale, flabby and softened. The lateral ventricles were very much dilated and contained a large amount of fluid. The medulla was flattened, compressed and softened.

Base of Brain.—Olfactory bulbs normal. Optic nerves small. Other nerves not examined.

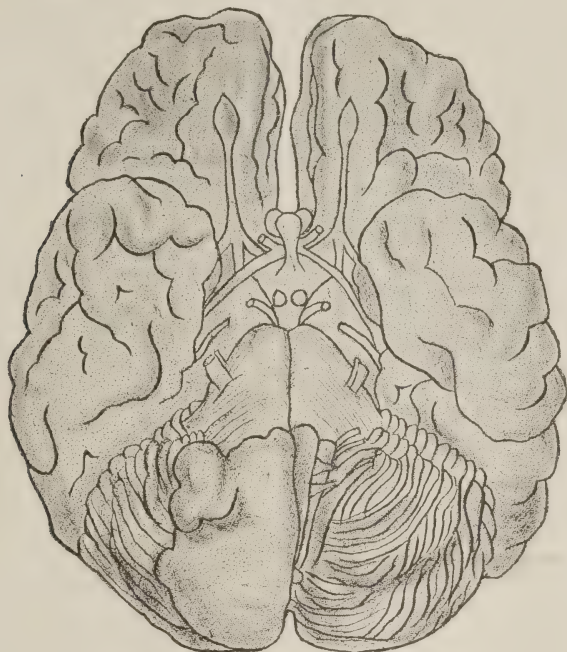


FIG. II.—Base of Brain. *T* points to Tumor in Situ.

Cerebellum.—Fig. 2. Placed directly between the lateral lobes of the cerebellum is a large nodular growth, three inches long, one and one-quarter inches wide, and one and

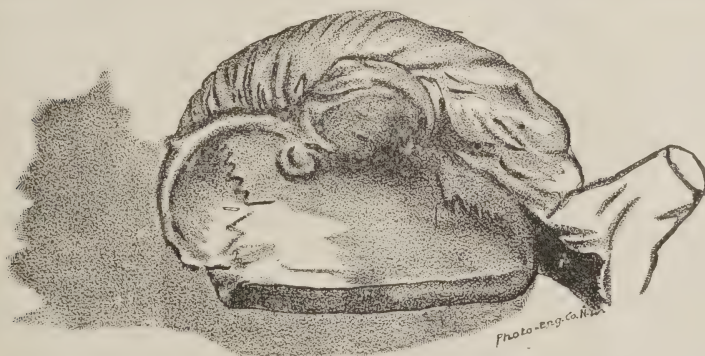


FIG. III.—Depression of Right Lobe of the Cerebellum, into which the Tumor fitted.

one-quarter inches in its vertical diameter. It extends into and is attached to the right lobe of the cerebellum, occupying quite an extensive hollowed out space in this lobe (Fig. 3). The growth also extended into the fourth ventricle, pushed the medulla to the left, and pressed on the right crus of the cerebellum.

Dr. Frank Ferguson makes the following report of the microscopical appearances of the tumor:

"The tumor is nodular in outline, in places cystic. Examination shows a large number of spindle cells, medium in size, imbedded in an abundant granular and fibrillated stroma, with a rich vascular supply. The walls, being composed of embryonic tissue, are quite thick and give the tumor the appearance of angio-sarcoma.

Society Reports.

PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, January 28, 1889.

THE PRESIDENT, S. WEIR MITCHELL, IN THE CHAIR.

Dr. F. X. DERCUM reported the following case of

SUPRA-DURAL HÆMORRHAGE.

The patient was a man, thirty to thirty-five years of age, brought to the Philadelphia Hospital early one morning in December by the police patrol, he having been found lying upon an open lot. He was totally unconscious; temperature subnormal, registering at one time 95°; face flushed and dusky; breathing labored and stertorous, and at times resembled the Cheyne-Stokes respiration. The lips and cheek of the right side were puffed in and out and the right nostril dilated. The left side seemed normal. The mouth was not distinctly drawn. Both hands and arms were firmly flexed and crossed over the chest. They were decidedly rigid. The legs were also fixed in extension with some turning in of the feet. The head was turned to the right, a marked effort being required to rotate it toward the opposite side. The right pupil was widely dilated, while the right eye was directed outward, as though held there by spasm of the external rectus. The left pupil seemed unaffected. Both were insensitive to light. No response was evoked by irritating the conjunctivæ, and this was also true of the skin over the entire body. The arms could be partially extended, and occasionally jerking movements could be observed in them.

The patient vomited freely, the ejecta smelling decidedly of whiskey. The urine, obtained by the catheter, contained

no albumen. The pulse was weak and intermittent. A small scratch or abrasion was found to the right and above the occipital protuberance. It had bled little, if at all, and seemed very insignificant. No bleeding had occurred from either the ears or nostrils. The man's condition remained unchanged for several hours, but he gradually grew weaker as the day progressed, and finally died.

Autopsy: Beneath the bruise previously observed the inner surface of the scalp and the periosteum were ecchymosed. No fracture of the skull was, as yet, apparent, and no depression existed. On removing the calvarium, however, and examining its inner surface, a fracture was detected beginning a little in front of the lambdoid suture and extending obliquely outward across the serrations of the latter. It was evidently a fracture of both tables, and on the outer surface was now observed to be about an inch in length and linear in shape. The fracture of the inner table was much more extensive. It extended in three directions, the most complete break being along the line of the lambdoid suture.

Lying upon the dura an immense clot was found upon the side of and beneath the fracture, and situated over the lateral aspect of the superior and inferior parietal lobules, the first and second temporal convolutions, and the anterior extremity of the lateral aspect of the occipital lobe. The anterior limit of the clot was about one-half to one inch back of the fissure of Rolando, while its posterior limit was from one-half to one inch from the apex of the occipital lobe. In the middle portion the clot was fully an inch, if not more, in thickness.

An examination of the base of the brain revealed numerous ecchymotic spots and areas in the pia and arachnoid. Similar appearances were observed in the brain almost everywhere. No hæmorrhage existed in the ventricles or ganglia. On the ventricular surface of the right caudate nucleus was an area of very superficial yellow softening one-half inch in length, one-fourth inch in breadth, and three or four lines in depth.

The fact of the limbs being rigid in contraction sug-

gested semi-conscious resistance, and also the fact that the man was vomiting matter smelling of whiskey, suggested that he was drunk. He undoubtedly was drunk in addition to having the extra-dural hæmorrhage. The diagnosis was also complicated by the presence of specific scars over his person, thus suggesting a specific cause for the inequality of the pupils. I, however, could not free my mind from a suspicion of hæmorrhage. I thoroughly examined the scalp, but did not feel at the time that the slight scalp wound was sufficient justification for trephining, but I think now that exploratory trephining would certainly have been rewarded. In explaining the symptoms in this case, it seems to me that the absence of hemiplegia was due to the fact that the clot was not directly over the motor area. The general rigidity is, probably, to be explained by irritation of the dura. The dilatation of the pupil was probably due to direct pressure.

Dr. CHARLES K. MILLS saw this man for a few minutes on the morning that he was admitted, and expressed the opinion that the case was one of hæmorrhage. The case is an important one, as it clearly confirms the diagnostic marks of Jacobson and others for supra-dural hæmorrhage. Looking over the cases which he had collected for the paper read at Washington, he found a number in which the symptoms correspond somewhat closely to those described to-night. The dilatation of the pupil—the oculo-motor paresis—is so common that Jacobson suggests that it be called Hutchinson's pupil, it having first been described by that observer. He examined the oculo-motor nerve very carefully in this case at the autopsy; it did not differ from the other nerves at the base. He, therefore, thought that the dilatation of the pupil was the result of pressure. A considerable amount of pressure would be exerted on the entire hemisphere, and a clot in the position indicated might possibly cause paresis of the oculo-motor nerve by counter-pressure. He believed that in a case presenting these general symptoms, with paresis on one side, and suspected injury, even without the external evidences present here, we would be justified in trephining over the parietal

lobe, as has been done by Kronlein and others in such cases.

HÆMORRHAGIC PACHYMENINGITIS.

Dr. FRANCIS X. DERCUM, in a series of seventy-seven autopsies, the records of which, and the specimens of membranes of which he went over very recently at the Insane Hospital at Norristown, found twenty-eight cases presenting hæmorrhagic pachymeningitis. In these twenty-eight cases there were four in which there were gross lesions, that is, they were large hæmatomata; in the others, there were fibrinous patches. Some of the patches were stained very slightly, while others were quite dense and heavily stained; all were more or less stained.

The frequency of the occurrence of this affection in the chronic insane is exceedingly interesting, and suggests that this hæmorrhagic or *fibrinous* pachymeningitis, as it is perhaps better called, is an inflammation, so to speak, normal to the dura—*i. e.*, if we have a simple inflammation of the dura, it is always an inflammation with fibrinous exudation, with or without the formation of vessels. Looking over the recent publication of Hirst on nervous diseases, he found that, under the head of inflammation of the dura, in which he is supposed to consider only simple inflammation, he speaks only of internal hæmorrhagic pachymeningitis, evidently considering this *the* inflammation of the internal dura, thus regarding all other inflammations as special forms.

Dr. A. ESHNER, through the courtesy of Dr. Hirst, reported to following case of

HÆMORRHAGE INTO THE CENTRUM OVALE OF THE POSTERO-FRONTAL AND PARIETAL LOBES.

E. L., an unmarried Englishwoman, aged thirty-three years, was admitted to the hospital December 18, 1888. She stated that she had been blind for three weeks. The ability to discriminate light remained. There was a moderate degree of anasarca, and examination of the urine disclosed a considerable amount of albumen, with hyaline and granular

casts of the uriniferous tubules. On December 20th the external measurements of the maternal pelvis were made, the diagnosis of pregnancy confirmed, and the presentation and position established. The foetal heart-sounds, however, were not detected. Upon the direction of Dr. Hirst, a hot-air bath was ordered, and the removal of the foetus, which it was conceived was dead, was contemplated. Shortly preceding the examination, on December 20th, at about five o'clock in the afternoon, the woman developed a comatose condition, with stertorous breathing. She had sat up in a chair during the morning, had taken milk and medicine, and, in response to inquiries, had stated that she was feeling better than hitherto. The night before, she is said to have sat up in bed and combed her hair, though a neighboring patient observed convulsive muscular contractions of the arms and shoulders. Soon after the condition of coma was noticed, the patient was yet able to swallow and respond to calls. She made complaint of the disagreeable taste of the medicament which she was taking. She was given milk again at 9.30 P. M. and at 1.30 A. M., and medicine at 10.30 P. M. Urine was passed once, and the bowels moved twice involuntarily. She grew worse during the night, and Cheyne-Stokes respiration set in. While preparations for a second hot-air bath were being made, at about 4.30 A. M., December 21st, the woman vomited a not large quantity of bloody fluid and expired. Unfortunately the nervous aspect of the case was not studied. It is known, however, that the patient was able to move the right upper extremity until within a short time prior to death. On the night before, she appeared to have been oppressed by a sensation of subjective heat, as she removed the bedclothing from the chest.

The autopsy was made under the direction of Dr. Mills, thirty-six hours after death. The only abnormal appearance on the surface of the brain was a large area of discoloration over the posterior part of the first and second frontal convolutions, at their junction, reaching in the same plane, over the two ascending convolutions. On opening the right lateral ventricle, an irregularly shaped, longitudinal clot, about one inch or more in length, was found lying

loosely in the cavity. This clot communicated with another, or with another portion of the same clot, which lay in a cavity which appeared to exist in the white matter external to and above the ganglia. Examination showed the ventricular surfaces of the caudate nucleus and optic thalamus entirely intact. The septum lucidum was not broken down, nor was there any blood in either the third or the left lateral ventricles. In fact, the entire left hemisphere appeared normal. A large section through the middle of the ganglia on right side and the parietal lobe revealed a clot of the size of a lemon, confined to the white matter of the posterior frontal and parietal regions—the white substance of the corona radiating between the ganglia and the following convolutions: the posterior portions of the first, second, and third frontal, both ascending, and the superior and inferior parietal. Transverse sections in various situations through the ganglia showed no lesion. The ganglia were intact.

Hæmorrhages into the central ovale and the cortex cerebri are relatively infrequent, though next in frequency of occurrence to those into the ganglia, which hold the first place.

Nothnagel, in Ziemssen's *Cyclopedia*, states that hæmorrhages into the medullary substance may be wholly unattended with evidences of cerebral disease. In some cases an apoplectic seizure may occur, followed by complete recovery. In others there may be the usual evidences of hæmorrhage, with or without apoplectic symptoms, followed by disorders identical with those following lesions of the corpus striatum. The symptoms of hemiplegia may be present and subsequently entirely disappear. This is perhaps the most characteristic feature of hæmorrhage in the region under consideration.

Vasomotor disturbances and alterations of sensibility are sometimes present. Contractures are rarely noticed. The symptoms arising from affections of the centrum ovale bear the closest resemblance to those produced by disease in the corpus striatum.

Gowers states that hæmorrhage into the centrum ovale

causes symptoms similar to those due to a cortical lesion in the corresponding situation, but without the symptoms of irritation. Large hæmorrhages usually spread into the centrum ovale from the corpus striatum. The vessels in the white substance are small and give only small hæmorrhages.

Dr. WM. OSLER said this case illustrates one of the conditions which in the pregnant woman may lead to hemiplegia or fatal hæmorrhage. True apoplexy occurs most frequently in association with Bright's disease, and may come on during, or even be mistaken for, a uræmic attack. A specimen very similar to the one here shown is in his collection of brains in the Museum of McGill College—massive intraventricular hæmorrhage. Rupture of a cerebral vessel during labor is, he believed, rare. A second more common event is embolism of one of the cerebral arteries during parturition. Aphasia is very often associated, and in a majority of the patients old mitral disease exists. In a limited number of cases a third condition is present—thrombosis of the cerebral vessels, due possibly to blood conditions which favor coagulation.

Stated Meeting, February 25, 1889.

THE PRESIDENT, S. WEIR MITCHELL, M.D., IN THE CHAIR.

Dr. S. SOLIS-COHEN read the notes of a case of

OBSTINATE SCIATICA CURED BY DEEP INJECTIONS OF
OSMIC ACID.

The method of treating sciatica by deep injections of a solution of osmic acid is so well known that the present case is reported only on account of its peculiar therapeutic history previous to the resort to the agent in question. Nearly every other medicinal and surgical means known had been tried faithfully, but without good result. The details of the case are given merely to show that osmic acid was really the curative agent.

George D., brakeman, æt. forty-five, was admitted to the Department of Clinical Medicine of the Philadelphia

Polyclinic, March 24, 1888. For twenty years the patient had had more or less pain in the lumbar region, of gradual development, attributed by him to the fall of a log upon his back while engaged in transport duty during the war. About thirteen months ago he began to feel pain in the right hip extending along the course of the sciatic nerve to the heel. The pain was constant with paroxysms of aggravation, very frequently preventing him from working or even standing erect. Sleep was continually disturbed. He had been under treatment first in the Department for Nervous Diseases, where the diagnosis of sciatica was made, and then in the Surgical Department, for about a year; having been subjected to the influence of a number of medicaments, including among many others, arsenic, potassium iodide, antipyrine, and antifebrin; injections of atropine, morphine, and theine; electrical treatment had been employed; and among surgical procedures, blistering, nerve-stretching by elevating the limb during ether anæsthesia, and, finally, nerve-stretching after incision and exposure. The latter operation was performed February 21, 1888. Immediately after the operation slight relief was experienced, although pain below the knee continued, and with the return of the power of motion in the limb, pain recurred as violent as ever; and had continued without intermission for about two weeks, except when the patient was under the influence of hypodermatic injections of morphine, without which he could not obtain sleep. He was then transferred to the general medical clinic. Salol being then under investigation, especially as to its analgesic properties, was prescribed tentatively in doses of about thirty to forty grains per diem. It gave relief, but not enough to warrant the hope of permanent good from its continued administration.

March 29.—An injection of ten minims of a one per cent. solution of osmic acid was made deeply into the thigh in the neighborhood of the point of emergence of the sciatic nerve, just above the cicatrix of the incision for nerve-stretching. On each of the two succeeding days, fifteen minims were injected. Improvement now began to

be manifested, although the pain continued to disturb sleep. Tri-weekly injections of twenty minims each were made during the next two weeks; a pill of morphine, belladonna, and quinine being given when necessary to produce sleep.

April 11.—The patient was improved sufficiently to sleep without the pill.

19th.—Improvement had steadily progressed, so that only a cane was used instead of crutches for walking. An injection of twenty minims was made higher in the buttock near the sciatic notch, pain being felt more especially at that point. Patient discharged from hospital.

May 17.—The patient returned, stating that he still had pain, coming on at twelve or one o'clock at night and lasting until morning. He was free from pain during the day, and could walk readily with the aid of a cane. An injection of thirty minims was made at the same point as previous injection.

January 1, 1889.—The patient being sent for, reports that after the last injection he was so sore that he could not sleep that night. The soreness gradually abated, and two weeks later he was able to sleep without interruption during the whole night. Since then he has had no pain of any account. There is some weakness of the leg below the knee, and some improvement in the movement of the foot, but not enough to interfere with locomotion with the aid of a cane. He considers himself well.

Dr. J. MADISON TAYLOR then called attention to

SOME POINTS IN THE TREATMENT OF SCIATIC NEURITIS.
MASSAGE AND INUNCTION WITH A GLASS ROD.

The remarks were mainly intended to call attention to a simple device which he has used with success in the treatment of sciatic neuritis.

Probably the best treatment for sciatica is absolute rest. This, coupled with the use of continued cold, with galvanism, the repeated use of the actual cautery, or blisters, usually does much good. But oftentimes more is needed,

in the shape of regulated exercise, passive exercise of the affected limb; in short, overstretching of the nerve by forced extension and flexion by a skilled manipulator, deep massage, and the like. These last seem often to do as much or more than the more radical measures, but require the services of an experienced assistant, which many cannot afford.

A year or two ago a patient, suffering from an obstinate form of syphilis, which resisted treatment at the best hands in this country, went abroad and got practically well in the hands of a clever physician in Germany, by the use of our ancient ally mercurial ointment, applied in a very novel and effective manner. This consisted of a glass rod, the size of a section of broom handle, two feet long, on which the ointment was smeared, and thence slowly and firmly rubbed into the skin. This combined the inunctions which could thus be made very thorough and systematic, with deep massage of the tissues. It certainly seemed to effect a more thorough introduction of the drug into the circulation, for the ointment was thus made to entirely disappear into the skin. The firm, slow pressure thus exerted must have an immensely stimulating effect on the muscles, nerve sheaths, etc., as well as on the absorbents, and is of itself an excellent tonic. This method he has not yet been able to test thoroughly in cases of syphilis, but he confidently recommends it as offering great possibilities of rapidly impressing the system with mercury.

Having a patient suffering from chronic sciatic neuritis, on whom the skill of several specialists had been wrought in vain, he determined to apply the same method of treatment. He used an ointment of mercury, belladonna, and iodine, applied on a glass rod, for fifteen minutes at a time. Very prompt relief from pain was felt. Later he resorted to the use of the rod without the ointment, and with apparently equally good results. In a short time, two or three weeks, his case was practically cured. Since then he has used this method in many cases, usually in the hands of the patient himself—a most convenient plan. Its use is followed by a marked sensation of warmth and comfort. In

one case, that of a gentleman who was obliged frequently to ride on horseback to his business out of town, the pain in both legs became intense at three or four o'clock in the morning, keeping him awake thereafter. Nothing so relieved this as ten minutes deep massage with the rod. In a month, pain which had resisted remedies for a year had gone.

Dr. CHARLES K. MILLS did not think he had exhausted all the remedies known to our art in the treatment of the case reported by Dr. Cohen; but he remembered that the patient was treated by various methods, medicinal and otherwise. This was one of several cases in which he employed hypodermic injections of theine. He believed that this patient received a large number of these injections. His success with this measure had been varying. In a few cases the patients had been benefited, but in the case reported to-night, and in others, the relief has been only temporary. The treatment of sciatica, like the treatment of chorea or rheumatism, is a subject about which we might talk for a long time simply enumerating the remedies that have been used. In his own practice he had a method of treating sciatica which, while it might not be scientific, was practical. He divided his cases into recent cases, old cases, and intermediate cases. Recent cases would commonly yield to such remedies as salicylate of sodium, oil of gaultheria, and analogous drugs, associating with this treatment the hypodermic injection of morphia and atropia, and sometimes of theine. For the old cases he used some very old remedies, and often with greater success than some of the newer remedies. He had used turpentine with success after the method first recommended by Thomas King Chambers. He also used Donovan's solution, in fair doses; also iodide of potassium and colchicum. In these cases he nearly always associated with the foregoing agents the use of cod-liver oil, massage, and electricity. On the whole, he got as good results from this treatment as from any other. He had also used the Adamkiewicz electrode, by means of which chloroform is applied externally and electrically. This treatment had proved successful in a few instances.

All, of course, had used counter-irritation. The use of the hot iron certainly acted better than any other form of counter-irritation. So far as osmic acid is concerned, he had had very little experience. He had used nerve-stretching by the method without cutting, and in a few cases by cutting, but never with any marked success.

The case reported to-night is valuable and interesting, but the report of a single brilliant success with a remedy like osmic acid in a case like this, does not carry the full force of conviction as to its great value. The disease had existed for a very long time, it is true, and it finally yielded apparently to this agent, not long after surgical and other procedures had been used.

Dr. JAMES HENDRIE LLOYD last year treated a case of sciatica which he cured in eight months, and he wished to refer especially to what seemed to do the most good. He first applied the hot iron very thoroughly; he burned four holes along the course of the sciatic nerve. This afforded some relief, but did not cure the case. The man was sick for at least six months after this application. During this time he employed the usual remedies, salicylate of sodium in large doses, antipyrine, etc. He packed the leg in flowers of sulphur for a time without benefit. He then had an exacerbatation of the pain and had to employ large doses of morphia hypodermically. The injections were made deep into the muscles, but he must have used a syringe which was not aseptic, for abscesses formed. These were quite troublesome and burrowed through the muscles of the buttock, but the effect on the sciatica was very marked. Improvement began as soon as there was a free escape of pus. He supposed that this acted simply as another form of counter-irritant. He however did not recommend this as a method of treatment.

Speaking of one case not establishing a principle reminded him that in the first year of his practice he saw an obstinate case of sciatica in the person of his preceptor. After it had continued six month, the application of the hot iron in exactly the same manner as he employed it in the case referred to, was attended with brilliant success. From

the time that he came out of the ether he never had a recurrence of the pain, and for the remaining two or three years of his life he had no return of the trouble.

Last year he saw removed, at the Philadelphia Hospital, a sciatic nerve, which evidently had been the seat of a good deal of inflammation. He could not help thinking that the proper way to treat such a case would be to cut down with antiseptic precautions and open the sheath of the nerve. He believed that if we could be positive that the nerve is in that condition, that would be *the* method which would give the best results. This would be only following out the old-fashioned practice, obtained from the Chinese, of puncturing the sheath of the nerve with gold needles. He did not believe that the rubbing of ointments on the skin, separated from the nerve by thick layers of muscles, could influence the inflammatory action. He was sceptical in regard to all such remedies.

Dr. F. X. DERCUM said that there was one practical point of importance in regard to the use of the actual cautery, that is, that the burn should be superficial, not deep. We want to impress the nerve-endings, not to make holes, and to heal up the burns as rapidly as possible. After applying the hot iron, dress the burn antiseptically and allow it to heal. It is, he thinks, the *repeated* burnings that accomplish the good. His ordinary method in the treatment of sciatica is to first use the salicylates. If ordinary doses do not give a good result, then to give as large a dose as the patient can bear. The suggestion of Dr. Lloyd in regard to opening the sheath, he considered a valuable one. If there is inflammation, incision of the sheath would allow the effusion to escape. Even if it did not accomplish good in this way, it might so alter the nutrition of the nerve that benefit would ensue.

Dr. S. WEIR MITCHELL desired to make a few remarks upon this disease, which is so often quite easy to treat, and again so excessively difficult. It has a host of remedies. It had been his own fortune to see in hospital practice an enormous number of cases of sciatica, and he really thought he had done something toward improving the methods of

treatment in old and obstinate cases. It is probable that a large number of slighter cases are of rheumatic origin, but when this condition endures for a long time, or there is a primary traumatic cause, it is pretty sure to end in inflammation of the sheath, or of the nerve track, or of both, with more or less effusion within the sheath. Much of this opinion may seem to be mere guess-work, for the opportunities of seeing the living nerve are rare. He had, however, in two cases during the war, seen the nerve cut down upon for the relief of serious traumatic neuralgia. In both there was a considerable amount of serum within the nerve sheath. In one, as he remembered, the nerve itself was distinctly swollen and inflamed. In both these instances four or more inches of nerve were exposed, and in both cases the results of the operation were excellent.

In speaking of the treatment of sciatica, he desired to say that if the case has lasted long, or has not, and is serious or slight, of course he need hardly refer to the need of the most careful attention to the constitutional condition of the patient; nor need he go into the consideration of the anæmic, scorbutic, or gouty states, which require and reward prompt treatment by internal remedies. He might say, however, that if these matters having been duly considered, in ordinary instances of sciatica, massage alone is often competent to do the rest. What Dr. Taylor had said in this connection is of interest. This mode of treatment by massage is what the French call *effleurage*, and not deep kneading. A roller of glass would be a good instrument for making this form of impression on the nerves. It is simple, and anybody could be taught to use it.

In more severe cases the cautery is his favorite remedy. He was perfectly at one with Dr. Dercum in reference to the manner of using it. It should be used so as not to destroy the skin, which ought to be touched lightly three or four times in places along the track of the nerve. This may be repeated every three or four days until a cure is effected. Such burns scarcely require any dressing. In children, or in nervous people, the previous application of

ice, or of ice and salt, will prevent any immediate pain. But as regards this and all local means of treating this disorder he felt quite sure that they are most successful when the patient is kept at rest in bed.

As to nerve-stretching, he had had it done for sciatica three times, and he had seen three cases in which it had been done, so that his whole experience rests upon six cases. He thought he had never seen a case cured by this means. He did not say that it did not relieve for a time, for it did. But he might also add that in the last few years he had seen cases of this disease treated by nerve-stretching with failure of relief, and had afterwards seen them cured by means which might have been used first. Strong extension of the muscles, such as involves more or less stretching of the nerves, he thought was of great value in certain spastic spinal affections. But he had never had any luck with this treatment of sciatica.

He would like again and again to repeat that in severe cases rest is an imperative need. By this he did not mean merely remaining in bed, but the use of methods which may or may not be altogether his own (that is a matter of indifference), but which he had never seen elsewhere described, and which are now often used in the Infirmary by himself and his colleagues. These consist in placing the patient who has this neuralgia in bed and putting on him an old-fashioned thigh-fracture splint, insisting on its being worn night and day, so that it is impossible for him to bend his leg. The result of this absolute rest is, in many cases, quite remarkable. Nor does this means forbid the use of other agencies, notably the cautery, or low temperature, or of other measures which may be allowable.

Another measure which he did not know to have been used by any other one than himself is the *persistent* application of ice along the nerve track. This consists in applying ice-bags along the whole track of the main nerve *in connection with the splint*; that is to say, these bags are applied from the sciatic notch to below the knee. They are kept on day and night. In one case they were used as long as three weeks, with triumphant results.

He had made some investigations in regard to the temperature obtained by these applications, and at a later date he would make a report on this, considering it to be a subject worthy of further investigation. He had used bags filled with ice, and had had ice-water circulating through the bag. He had found that, by the use of different saline solutions with ice, it was possible to secure certain definite temperatures in the skin under the bags.

Dr. WHARTON SINKLER desired to call attention to one method which had not been spoken of. That is the method of acupuncture by the German apparatus called Baumscheidtmsus, consisting of a number of needles which are driven into the skin in the course of the nerve and some counter-irritant oil rubbed in. He had cured one case of most obstinate and violent sciatica by this means, and the patient remained well for a number of years. In this case, however, it is possible that the result was influenced by the sloughing which occurred at the point of puncture. In another case, in which sloughing did not occur, a cure was also effected.

Dr. S. SOLIS-COHEN was aware that one case does not establish a general rule, and on that account simply reported this case without comment. Osmic acid, however, is not an untried remedy. There are very many cases of its use on record, principally during the last five or six years. He had used it in some twelve or fifteen cases, but only two were in private practice, and the notes in regard to the others are not at present accessible. His impression is that the result was satisfactory in about one-half of the cases. The German physician who first investigated this method reported upward of forty or fifty cases, and found that the results were best in the oldest cases. It was useless in the acute cases. Some experiments on animals were reported, showing that a definite alteration in the nerve cells, similar to that observed in histological preparations treated with the same agent, followed the injection of the remedy. It is probable that conduction is interfered with. He did not know how else we can explain the good result. He agreed with the speakers who consider internal

remedies, except where there is a constitutional or diathetic fault, as useless, except for palliation. Where the sciatica depends upon gout, or rheumatism, or anæmia, of course, salicylates, iron, arsenic, etc., as indicated, will often effect a cure, or greatly assist recovery. The value of iron specially needs emphasis, not only in anæmic but certain rheumatic conditions. Among remedies not mentioned, he had seen good results from the deep injection of chloroform. He had also occasionally seen temporary good results from the deep injection of atropine and morphine, in cases where the injection of morphine alone was without curative effect. The long list of agents and methods, each of which has been useful in some instances, shows there is no single treatment; but he believed that osmic acid, deeply injected near the seat of greatest pain, is among the remedies eminently worthy of consideration in unyielding cases such as that reported.

Dr. J. MADISON TAYLOR said that there is an anatomical point which he did not see credited with the influence which it deserves in determining the frequent occurrence of neuritis of the great sciatic near its exit from the notch. The nerve is very exposed just here, and it suffers direct compression by the action of several powerful muscles which run directly transverse to its course. The piriformis alone acts as almost a constricting band in front, opposed by the obturator interni and Gemelli beneath. In whatsoever attitude the body assumes when any of these muscles are put in tension, there is strong compression exerted on these tender fibres. Hence, when inflammatory action occurs in the nerve or its sheath, it is brought to a focus here, the circulation is interfered with, and the smallest muscular movements cause pain and irritation. It is seldom granted us to use this organ in its earlier subacute or chronic condition of engorgement and thickening, but it is pretty certain we should find a red, swollen organ, with a jelly-like or sero-fibrinous exudate, crowding the sheath space, with here and there bloody extravasations. Now, next to the actual removal of this diseased section, as suggested by Dr. Lloyd, but not likely to be yet generally adopted, to his mind,

comes the gentle systematic compressing action by such means as he had suggested, which aids in breaking up old adhesions, and emptying out the engorged sheath, and such other results as aid a re-establishing of the normal elasticity of the parts. This massage does, but no hand can beat the power which this simple agent possesses, nor does the percutor of Mr. Mortimer Granville, so excellent in its results, do it any better, and not nearly so cheaply.

Dr. J. CHALMERS DA COSTA reported

FOUR CASES OF COCAINE DELIRIUM,

two of which occurred in his office practice, and two were observed in the surgical out-patient department of the Jefferson College Hospital, and the latter he is enabled to report by the courtesy of Dr. Horwitz, the department chief.

CASE I.—Man, twenty-five years of age, of nervous temperament, and addicted to the excessive use of alcohol. He had a stricture of the urethra, and had been for some time under treatment, but as the passage of instruments occasioned considerable pain, it was determined that cocaine should be employed.

He was handed a syringe containing M xxx. of a four per cent. solution, and told to throw it into the urethra. In a moment, on turning, he discovered him still seated on the edge of the sofa, one hand grasping the penis, the other the syringe, but pale and immobile as a statue; he spoke to him, but he made no answer, and when he touched him he fell over. He did not seem to breathe; the pulse at the wrist was imperceptible, the pupils were widely dilated, the lips were pale, the face pallid and bathed with sweat; there was muscular resolution, and complete unconsciousness. In about fifteen seconds breathing began, and the pulse reappeared. The pulse beat fifty to the minute, was small, and of high tension. The respirations were shallow and slow. The pupils were moderately dilated, and did not repond to light. Muscular resolution and unconsciousness were complete. Conjunctival reflex was abolished.

The extremities were icy-cold. There was entire insensibility to pain. The body was drenched with sweat. After several minutes, the face showed signs of venous congestion, the facial muscles twitched convulsively, the arms and legs were worked in a violent and irregular manner, and the patient tossed about on the sofa. The conjunctival reflex returned, but there was still analgesia. He soon began to talk incoherently, to laugh, shout, and sing, but paid no attention to words, or to being roughly shaken, and seemed entirely oblivious to his surroundings. He obviously had hallucinations of sight and hearing of an agreeable nature. The pulse was now rapid, weak, and irregular. The respiration shallow, frequent, and jerking.

This delirium gradually passed away; he would answer when spoken to, but could not maintain a thread of conversation, and when left to himself was concentrated on his own ideas, which flowed in a torrent, now grave, now gay, now majestic, now amusing. This condition was one of intellectual brilliancy. He quoted poetry, oratory, and philosophy (being a particularly well educated man, and a writer himself of some attainments). He gave portions of "Locksley Hall," with excellent effect, and wept as he recited Keat's "Ode to a Nightingale." When told that he had fainted while grasping his penis, and asked what would have been said by his friends had he died in that attitude, he responded, "The ruling passion is strong in death." He said he felt "gloriously happy," and that "a drowsy numbness filled his veins." Gradually he became quiet, and about one hour after the onset of the symptoms passed into an apparently natural sleep from which he awoke in two hours, feeling languid and somewhat giddy; complaining of headache and numbness of the extremities, dryness of the mouth, dimness of vision, and an intense desire to make water.

He remembered having begun to take the injection, but could recollect nothing more until he had reached the stage which he denominated a "poet's dream." In this stage, he says, his happiness was complete; he felt no care, took no note of time, experienced no surprise as to his situation,

and was occupied with poetic thoughts, dressed in splendid imagery.

He states that this mental condition was apparently identical with that occasioned in him by large experimental doses of hashish.

This gentleman, being of an experimental turn of mind, repeated the injection one night, with the result of alarming his family to a terrible extent. The family physician, who was sent for, expressed a strong and angry suspicion that alcohol was the whole trouble. The after-effects of this dose were decidedly unpleasant, and lasted many hours; being chiefly great prostration, numbness of the extremities, dry mouth, and dimness of vision.

CASE II.—Man, twenty years of age, brother of the preceding, of similar habits and temperament. Had been for some time under my care for urethral stricture. On his solicitation, M xv of a three per cent. solution of cocaine were injected into the urethra. Several bougies were passed without giving any pain. Some five minutes after the injection it was observed that his attention was withdrawn from the operation, and that he gave no answer when spoken to. The face was pale and covered with sweat, the pupils were moderately dilated, the pulse slow and of high tension, the respirations rapid, jerking, and irregular. Conjunctival reflex was not abolished, and on testing it he began to roll round, to toss his extremities, to shout and swear. He cried out that he could not breathe; he had hallucinations of sight, brushing bugs off his coat; and illusions of personality, thinking I was his brother.

He arose from the couch, staggered like a drunken man, and incoördinately adjusted his garments. His face was now flushed, and his pupils of normal size. He staggered about the office, upset chairs, aimed blows at me, and, with indistinct articulation, declared that I wanted to kill him. He insisted on going out, was with great difficulty got into a carriage, taken home, and put to bed, where he finally went to sleep, under sodii brom. and morphia. He awoke in the morning feeling as if he had been "on a tear," and

dimly remembering that he had abused me violently, for which he was very penitent. He remembered coming to my office, but had no memory of taking the injection, or of being operated upon.

CASE III.—Man, twenty-two years of age. Also a drinker. Came to the hospital to be operated upon for phimosis. 10 ℥ of a four per cent. solution of cocaine were thrown into the prepuce by means of a hypodermatic syringe. The operation was not entirely painless. About fifteen minutes after the injection he became very pale, and tetanically rigid, like a person in the first stage of an epileptic fit. The pupils were dilated, face was pale and covered with sweat, extremities cold, and he was unconscious. After a few seconds he tossed his arms, body, legs, and head, in a manner suggestive of the progressive movements of hystero-epilepsy. The pulse was rapid (120), weak and irregular. The respirations shallow and rapid. The reflexes were dulled, but not abolished. Sensibility was diminished, but not destroyed. He talked rapidly, inarticulately, and incoherently, prayed, swore, threatened, and tried to get up. In about twenty minutes he became quiet, was put to bed in the wards, and after some hours went home. Next day felt all right.

CASE IV.—Man, thirty years of age. Nervous temperament. Habits unknown. Came to the hospital for urethral stricture. Passage of instruments was productive of so much pain that 20 ℥ of a six per cent. solution of cocaine were injected into the urethra. In a few minutes he began to mutter, and to move his right hand as if sending a telegram (he was a telegraph operator). He began to talk about the message, saying it must be sent, and telegraphed with tremendous energy. His face was pale and moist, pupils of normal size and reacted to light, conjunctival and plantar reflexes present. Did not answer when spoken to, and did not feel a pin point. Respirations were rapid and shallow. Pulse was slow, and of considerable tension. This condition existed for some ten minutes, when he became conscious, and asked what was the matter. He complained of dyspnoea, and had no memory of events after the passage

of the first bougie. Next morning he stated that he felt dull and heavy for a number of hours after going home, that he found great difficulty in picking up small objects (pens, pencils, etc.), and that his fingers seemed numb, and could scarcely feel objects or grasp them.

Some physicians who have seen cases similar to these have stated their belief that the symptoms were purely the result of fright; but only one of these cases showed any apprehension.

It might be considered possible that such phenomena could ensue from a condition of reflex irritation, similar to the so-called pleuritic epilepsies which occasionally result from injecting the pleural cavity with iodine or some other agent. It seems improbable, however, that this was the condition here, as previous instrumentation on three of the cases induced no reflex disturbances and a repetition of the injection in one reproduced the symptoms.

He asked if the high arterial tension, the small and usually slow pulse, the pallor, the dilated pupils, and the unconsciousness are not evidences of cerebral anæmia from vaso-motor spasm. An interesting fact of this series of cases is the exhibition of a family idiosyncrasy by the occurrence of toxic symptoms in two brothers.

He inquired if the nervous temperament and the abuse of alcohol did not render the organism particularly susceptible to the action of cocaine, and if any signs exist which would indicate to us that cocaine would be dangerous in a given case.

Dr. FRANCIS X. DERCUM had repeatedly given cocaine in half-grain doses by subcutaneous injection without noticing any serious symptoms. This report is exceedingly interesting, and it is a question in his mind whether or not the locality in which the cocaine was applied had anything to do with the symptoms. The urethra is very sensitive and is intimately associated with the spinal centres. We must take this into consideration. It seemed to him that the case could hardly be considered proved unless a cross-experiment was made by injecting a similar dose of cocaine into the subcutaneous tissues of the patients whose interesting symptoms had just been detailed.

Dr. MITCHELL thought that the point in regard to the location of the injection was well taken. He knew by personal experience that different methods of taking a drug produce very different effects. When he himself takes morphia by the stomach, it produces horrible vomiting and headache, but when taken hypodermatically no inconvenience is produced; but this is common knowledge. What has been here told as to the occasional effects or the apparent effects of cocaine, when used by the urethra, should lead to some examination into the matter, since it is possible that there may be peculiarities due, in certain constitutions, to local impressions made on that sensitive tube by drugs.

Dr. LLOYD asked what is the proper dose of cocaine. He was using in one case two grains daily in divided doses, so far without any apparent effect.

Dr. E. N. BRUSH had had more or less experience with the injection of cocaine when it was first suggested for the treatment of certain forms of melancholia with stupor. He employed it in doses from one-half to one grain. The effect was to induce laughter in some, and a talkative state; it was only temporary and no serious symptoms were produced.

Personal experience had taught him that the effect of this drug on certain persons, even in good general health, is to render them more talkative. Last fall he used a four per cent. solution as a spray for a throat trouble, continuing the application but a few minutes. A talkative state was produced and in twenty minutes there was severe headache. He repeated the experiment on other occasions with the same result. He had had under his care a physician who, when he came to the hospital, was taking ten grains of cocaine daily. He had taken as much as thirty grains, he cut him down to two grains, then to one grain, and then stopped it altogether. There was no disturbance. He had had in all under his care three physicians who were addicted to the use of cocaine. Two used it hypodermatically and one by the mouth. The latter took forty grains a day, according to his own statement.

Dr. J. CHALMERS DACOSTA said that in hastily looking over the literature he found a very wide range or variation in reference to the dosage of cocaine. Martindale states in his book on coca and cocaine, that one man took without obvious ill-effect twenty-three grains. Another took, with suicidal intent, thirty-two grains, the result being considerable prostration. Dr. Dien, of Dantzic, an ophthalmologist, injected one-tenth of a grain under the conjunctiva and produced very alarming symptoms. The dangerous dose therefore seems to be somewhere between the wide extremes of one-tenth of a grain and thirty-two grains, but exactly where it is hard to tell.

PERISCOPE.

PHYSIOLOGY OF THE NERVOUS SYSTEM.

By DR. CARLOS F. McDONALD

INCREASING FREQUENCY OF GENERAL PARALYSIS OF THE INSANE.

Dr. H. Rooke Ley, Medical superintendent of the Prestwick County Lunatic Asylum, England, in the report of that institution for 1888, says :

“The type of insanity has of late years markedly changed, and many kinds of mental disorder, such as epilepsy, general paralysis, and its allied forms of structural brain disease, which may be termed incurable from the day of their development, are more common now than formerly. Insanity, associated with epilepsy, such as is met with in asylums, is nearly always incurable, and general paralytics are a class who usually go from bad to worse. The statistics of this asylum show that the proportion of patients suffering from organic brain diseases among the admissions have more than doubled within the last twenty years. Nearly twenty-five per cent. of the male admissions of 1888, were cases of general paralysis, a disease which, while comparatively unknown in Ireland, Scotland, and among rural populations generally, is prevalent in an increasing extent in Lancashire, Middlesex, and elsewhere where the proportion of urban population preponderates. It would appear as if the general tendencies of civilization, in large cities and populous manufacturing districts, are such as to exhaust nervous vitality and predispose to structural nervous degenerations. The Irish peasant, in his native country, has a marked immunity from these fatal forms of brain disorders, but

when transplanted into centres of labor and activity in Lancashire or Middlesex, he is often apt to break down and acquire a form of mental disease, progressive in its nature, and little susceptible of cure. So increasing is this form of insanity among the inhabitants of crowded communities that the recovery rate, in an asylum drawing its supplies from these centres will, no doubt, be seriously modified by it in future. This disease is principally confined to the male sex, but of late years has become more prevalent among women, no less than twenty-six, or six per cent. of the total female admissions last year were suffering from that fatal form of brain disorder."

It is with great pleasure that we announce the appointment of Dr. Carlos F. McDonald, Medical Superintendent of the State asylum for insane criminals at Auburn, New York, to the chair of Mental Diseases at Bellevue Hospital Medical College.

Dr. McDonald has for years been one of the co-operators of this Journal.

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A STUDY OF EXPERIMENTAL MYELITIS.¹

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THE object of this article is twofold; first, to show that it is possible to give rise to an acute localized myelitis in animals by shutting off, temporarily, the blood supply of the spinal cord; secondly, to describe with some detail the histological characters of the process thus induced.

The basis for the statements contained in these pages is a series of seventy observations, made chiefly upon rabbits, in the Pathological Department of the John Hopkins University, under the direction of Dr. Welch, during the winter of 1887.²

The method employed to deprive the cord of its blood supply was the old Stenson experiment, as modified by DuBois-Reymond. This consists in passing a curved needle through the abdominal wall on one side of the vertebral column and bringing it out on the other, in such a manner as to include, among other structures, the abdominal aorta and vena cava. The needle carries a strong ligature, which is tied so tightly over the back of the animal as to effectually compress the abdominal vessels. The

¹ Read before the New York Neurological Society, Feb. 5, 1889.

² Though written in 1887, no alterations have been made in the text.

spine of the fourth lumbar vertebra is the level at which the needle is passed.

The effect of this proceeding is to produce an almost instantaneous paraplegia; but precisely how it cuts off the circulation from the lower end of the cord, it is not possible to say without making a careful study of the blood supply of the cords of such animals as are used for experiment. In rabbits the arterial supply of the lower end of the cord closely resembles that of man in principle.³ There is a vertical continuity of the vessels of the cord, but it is probable that the direction of the circulation is chiefly horizontal.

That the Stenson experiment does in great measure deprive the extremity of the cord of its blood, and that the vertical anastomoses referred to only inadequately replace the original blood supply, can be shown by a simple experiment. The abdominal vessels are compressed in the manner described, and the vertebral arches are carefully removed from the fourth lumbar vertebra down to the coccyx. The cord is now divided transversely at about the level of the seventh lumbar vertebra, and the cut surface of the upper end is examined. The vessels of the pia mater are empty and the central gray has a dull ashen color.

If now the ligature which compresses the abdominal vessels be slightly loosened, a distinct change is at once noticeable in the cord. The vessels of the pia mater become filled with blood and show distinctly, and the gray matter grows brighter in color, although no decided change can be detected in the hue of the white substance.

When the ligature is again tightened the vessels of the cord once more become anæmic.

This effect of the ligature upon the cord is noticeable as high as the fifth lumbar segment. At the level of the first lumbar segment the circulation appears to be uninfluenced by the compression of the abdominal vessels. These facts

³ There are two anterior spinal arteries connected opposite each segment of the cord by a short transverse vessel.

are significant when we consider the character and limits of the cord lesion.

I have remarked that paraplegia follows compression of the vessels and is almost instantaneous in its onset. If the compression be exercised for a shorter time than three-quarters of an hour, the paralysis wears rapidly off, and in cases of this kind the paralytic effect is doubtless referable to the temporary ischæmia of the gray substance of the spinal cord.

If, however, effectual compression be continued during a period greater than one hour, the paraplegia becomes a permanent condition, and does not diminish during the life of the animal. In such instances lesions of the spinal cord can always be detected soon after the operation, as was pointed out in 1884 by Ehrlich and Brieger.⁴

These observers published their results in the form of a preliminary and somewhat fragmentary communication, in which they described lesions not only in the spinal cords of animals which had been subjected to the Stenson operation, but in the peripheral nerves and muscles as well.

In the cords of animals which had survived the operation a considerable length of time, they observed a separation of the gray substance from the white, with destruction of the ganglion cells and medullated nerve fibres of the anterior horns, and eventually also sclerosis of certain white tracts, particularly the short tracts of vertical association-fibres.

In the sciatic they found occasional hæmorrhages, and after the third day subsequent to the operation a degenerative process, similar to that which occurs in the peripheral portion of a cut nerve.

Near the junction of the paralyzed muscles with their tendons, sharply limited areas of coagulation necrosis were sometimes seen, and in advanced cases there was a considerable increase in connective tissue.

⁴ Ueber die Ausscheidung des Lendenmarkgrau. Zeit. f. Klin. Med., Bd. vii., Suppl. (84).

My own results bear a general resemblance to those of Ehrlich and Brieger, but differ from them in many important particulars, to some of which I shall allude later.

Before describing the lesions which occur in the spinal cord, and secondarily in the nerves and muscles, I wish to give a brief account of the clinical history which follows the Stenson operation; a clinical history closely resembling that of certain forms of acute atrophic paralysis in man.

In the great majority of instances the onset of the paralysis is so closely connected in time with the tightening of the ligature, that it may be considered coincident with it. In some cases, however, several seconds pass before the paralysis is evident, and occasionally the time is several minutes.

The effect upon the bladder and rectum is not always so rapid, but in most cases the paralysis of the sphincters of these organs is soon made evident by dribbling of urine and escape of fæces. Generally this sphincteric paralysis wears away rapidly, but the detrusor urinæ is permanently paralyzed and permits the accumulation of urine. The bladder thus becomes gradually distended, until a condition is reached in which there is a more or less continuous dribbling of urine. Alkaline fermentation occurs in the retained urine, and evidences of cystitis are soon present. Unless the bladder be emptied by compression from without, or by catheter, excoriations, ulcers, and fissures of the skin soon appear upon the parts over which the urine passes.

The paralyzed muscles of the legs remain relaxed for about two weeks, at the end of which time contracture sets in. The muscles most affected are the extensors and adductors of the thigh and the flexors of the leg, so that in course of time the lower extremities become fixed near the body.

Fibrillary contractions sometimes occur in the course of a few weeks in the contracted muscles, and may continue at intervals during the life of the animal.

Slight atrophy of the muscles of the thigh and leg is usually perceptible at the time the contractures begin, i. e.,

about the end of the second week. By the end of the fourth week the affected muscles are usually very much reduced in size, in some instances to one-half or one-third their original bulk.

I have made no satisfactory observations with regard to the electrical reactions. Erlich and Brieger, however, state that the nerves derived from the diseased region of the cord remain excitable from twenty-four to thirty-six hours after the removal of the ligature. By the end of the third or fourth day their irritability is permanently lost. The muscles remain excitable to electricity and to mechanical stimuli until rigidity sets in.

Sensation does not appear to be affected. At all events, it is not perceptibly altered during the first four weeks. Painful stimuli to the skin of the paralyzed legs are resented, and the animals at times cleanse the affected parts with their tongues, an act probably suggested by the disagreeable impressions to which the presence of dirt gives rise.

It is probable that at the end of the first month, sensation may be to some extent modified. As will be seen later, the posterior nerve roots are at this time beginning to undergo degeneration, and the altered sensation and any trophic changes which might exist at this period could thus be accounted for. In the only animal which lived just one month it was impossible to say with certainty that sensation was modified, or that the falling out of the hair which occurred was not due to the action of decomposing urine.

The tendon reflexes seem to be lost in every case after the third day.

Diarrhœa was present in about two-thirds of the cases. It came on soon after the operation, and its duration was usually about forty-eight hours, sometimes only a day, sometimes a week. In a few instances second and third attacks of diarrhœa occurred. Why it should occur at all, I do not know.

Immediately after the cutting off of the blood supply to the cord by the tightening of the ligature, the temperature falls. During the first ten or fifteen minutes there is a fall of one, two, or even three degrees Fahr. In the course of an

hour there is an additional decline of one or two degrees, and after the lapse of four or five hours the maximum fall, which is frequently a drop of six, seven, or eight degrees from the normal, is reached. The temperature then remains stationary for several hours, and if the animal is to recover from the shock of the operation, the temperature begins to rise very gradually, reaching the normal point in about twenty-four hours after the removal of the ligature. In the rectum and in the parts deprived of blood, the temperature falls more rapidly than in the surrounding region, but eventually a nearly even temperature is reached throughout the body.

In a few cases the initial paralysis soon passed away almost completely, leaving only slight paresis of certain muscles. Contracture has set in, in these cases, in the same manner as when the legs were permanently and completely paralyzed.

In a very small proportion of the animals operated upon, only one leg was paralyzed and the bladder and rectum were unaffected. The clinical history in these cases closely resembled that of the monoplegic type of acute poliomyelitis.

Paraplegia, identical with that produced by Stenson's experiment, can be brought about by simply tying the abdominal aorta below the origin of the renal arteries. The Stenson operation is however the preferable method, as it is very difficult to obtain recovery in animals in which the abdomen has been opened.

I have examined the spinal cords, nerves, and muscles of rabbits in which paraplegia has been induced, at periods of time varying from a few hours to thirty days after the operation, and upon these examinations the following description of the lesions is based.

Spinal Cord.—The first alterations in the structure of the spinal cord were confined to the ganglion cells of the anterior horns at the level of the fifth and sixth lumbar segments. In sections made from this region of the cord thirty-six hours after operation, a few ganglion cells were present in which the cell-body was pale and swollen, and

in which the nuclei were obscured. Twelve hours later a larger number of cells was involved. As before the cell-bodies were pale and swollen, but in addition many of them had lost their processes, and in many instances the nuclei were absent. No other changes could be with certainty made out.

After the lapse of seventy-five hours the necrotic changes in the ganglion cells had increased in intensity, and had involved, at least to some extent, about one-half the cells in each section. Many of the ganglion cells nearest the centre of the gray substance were shrivelled, granular, and without nuclei; some were hardly recognizable as the débris of dead cells. The majority of the normal ganglion cells occupied the periphery of the gray substance, and between these groups and those centrally situated, cells could be seen in all stages of destruction.

In consequence of necrotic changes in certain ganglion cells, some of the nerve fibres of the anterior nerve-roots were involved. In some cases the medullary sheaths and axis-cylinders had entirely disappeared. In other instances axis-cylinders were left naked by the disappearance of the myeline sheaths.

Sections successfully stained by Weigert's method, showed that the fine network of medullated fibres of the gray substance had been distinctly compromised. Many of the fibres were badly broken up, segmentation of the myeline had occurred, and many naked axis-cylinders were visible. These changes were most marked near the centre of the anterior horns.

Immediately about some of the capillaries of the gray substances there seemed to be a slight increase in the number of nuclei, but whether this was sufficiently marked to be considered pathological I cannot say. Hæmorrhages into the gray substance were always present at this stage, although it is stated by Ehrlich and Brieger that they never occur. They may indeed be found twenty-four hours earlier. These hæmorrhages were neither numerous nor large, and were frequently located near the centre of the anterior horn.

Hæmorrhages into the pia mater, and between the pia mater and the cord are numerous, and are apt to be large. The vessels of the pia are large and filled with blood cells.

When six days have passed several modifications of the condition just described are noticeable, and certain new changes make their appearance. A much larger number of ganglion cells is now involved in the destructive process, and only those occupying the periphery of the anterior horns remain unaltered. A few have been entirely destroyed, and others are greatly atrophied. As before, the cells most altered lie in general near the centre of the anterior horn.

The arterioles and capillaries of the gray substance are dilated and filled with blood cells, and in the former the nuclei of the endothelium appear to be increased in number. There are probably no newly formed vessels.

Immediately about the blood-vessels, and particularly about the capillaries, are accumulations of leucocytes, constituting border-zones. This formation of border-zones begins about the fourth day, in the fifth and sixth lumbar segment, and is a well established condition at the time of which I am now writing. These foci are very numerous, particularly near the centres of the horns, and vary considerably in size.

The changes in the anterior nerve-roots are of the same character as before, but involve a greater number of fibres. In addition a slight increase in nuclei along the borders of the nerve-roots is sometimes present. This is the precursor of the infiltration which occurs subsequently. The posterior nerve-roots are normal.

The network of medullated fibres is more extensively damaged now than at any previous time, and the destruction is particularly noticeable where the leucocytes have gathered. The medullated fibres, however, which contribute to the formation of the posterior nerve-roots, are not at all or only slightly damaged.

Sections made through the fifth and sixth lumbar segments show an interesting and rather extraordinary altera-

tion in the central canal of the spinal cord. In the cords of normal rabbits the shape of the canal is commonly elliptical on transverse section, the long axis of the ellipse being antero-posterior. In the sections referred to, the canal is transformed into a long narrow slit, the long axis of which lies at right angles to the normal direction.

In the interval between the anterior and posterior borders of the canal lie a few leucocytes and some granular material. The lining epithelium is generally normal. Why such a remarkable change in the contour and direction of the canal should occur it is difficult to imagine. It is possible that the diminution of consistency in the anterior horns on either side of the central canal, and the intracanalicular pressure, may be mechanical factors in the production of the condition.

The vertical extent of the lesion is at this period considerably greater than before, the extension being particularly in a downward direction. Evidences of the inflammatory process can be detected as high as the middle of the fourth lumbar segment, and as low as the lower end of the sacral cord, but the fifth and sixth lumbar segments are more seriously involved than any other portion of the cord.

The transition from the diseased cord to the normal structures above it is gradual. The upper limit of the lesion affects a conical mass of the gray substance of each anterior horn, and the higher we examine above a certain point the smaller is the number of diseased cells. At length only one or two of the more centrally situated cells of each horn are diseased, and finally perfectly normal tissue is reached.

Most of the changes mentioned as occurring in this stage of the destructive inflammation are grossly illustrated by Fig. II.⁵ Comparison with Fig. I. will serve to contrast the pathological and normal conditions.

⁵ All of the figures are of a semi-diagrammatic character. The small figures of the spinal cord are intended to give a general idea of the topography of the lesion.

During the three days following this stage the changes in the cord continue to increase in intensity and to widen in extent.

When nine days have passed, the foci of small round-celled infiltration are so numerous in the fifth and sixth lumbar segments, that they coalesce to form areas of considerable size. The infiltration is most dense, and the infil-

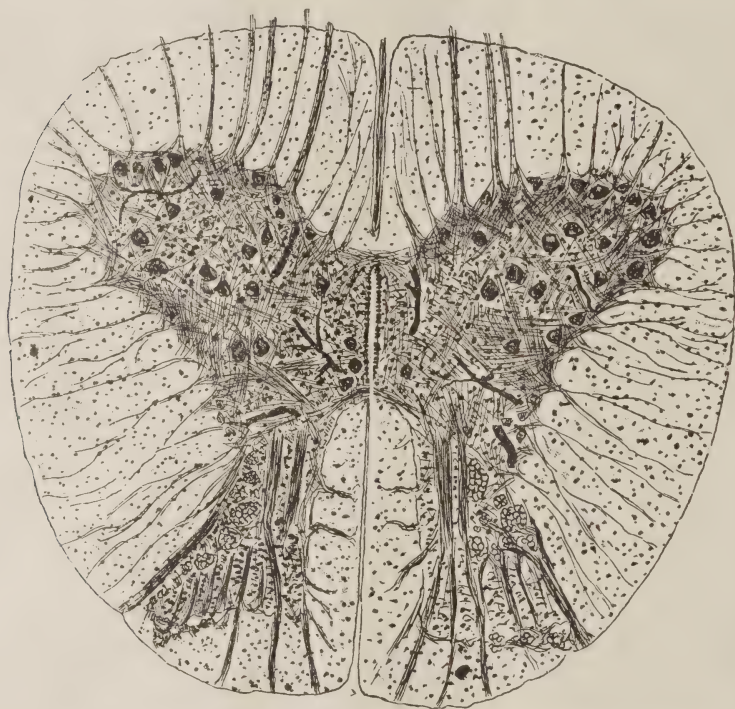


FIG. I.—Section through normal cord of rabbit, at fifth lumbar segment. Semidiagrammatic.

trated areas are largest near the centre of the anterior horn. A considerable number of leucocytes are present in the substantia gelatinosa of the posterior horns, but the posterior nerve-roots are normal.

The anterior nerve-roots are in very much the same state as at the end of the sixth day, except that there is now a very distinct infiltration of small round cells, into the connective tissue of the sheaths of the roots. Leucocytes

are also found in considerable number in rows along the borders of the roots, and in the space previously occupied by the fibres. A few ganglion-cells, apparently normal, can usually be detected along the anterior border of the anterior horn, even in sections from the region of the cord most compromised.

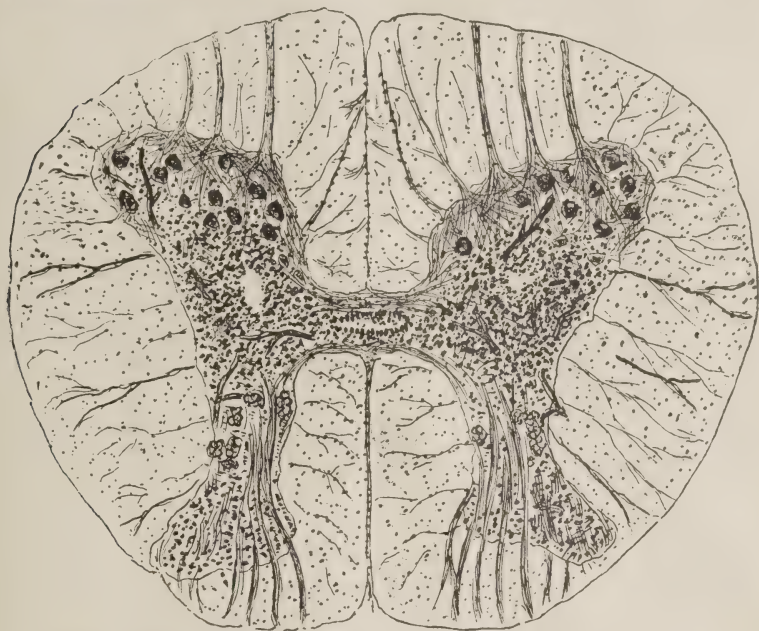


FIG. II.—Section through fifth lumbar segment of cord, six days after operation. Destruction of centrally situated ganglion-cells. Accumulation of nuclei. Change in direction of central canal. Anterior nerve-roots only slightly altered. Post roots intact.

The condition of the central canal is the same as at the sixth day. The change is present, however, through a greater vertical extent of the cord.

A rather curious separation of the gray substance of the anterior horns from the white substance immediately surrounding it, is evident to the naked eye at this time. The separation is often so complete and sharp that the anterior horns literally fall out of sections made through the most diseased region. In the middle of the lumbar cord this condition begins to be evident several days before the time

of which I now speak. Eventually it is present, at least to a slight extent, throughout the affected region of the cord. The separation is doubtless due to the breaking down of tissue along the edge of the horns, in consequence of the accumulation of leucocytes.

When twelve days have passed no normal ganglion-cells can be found in the fifth and sixth lumbar segments. Only a few swollen granular cells, lying generally near the exit

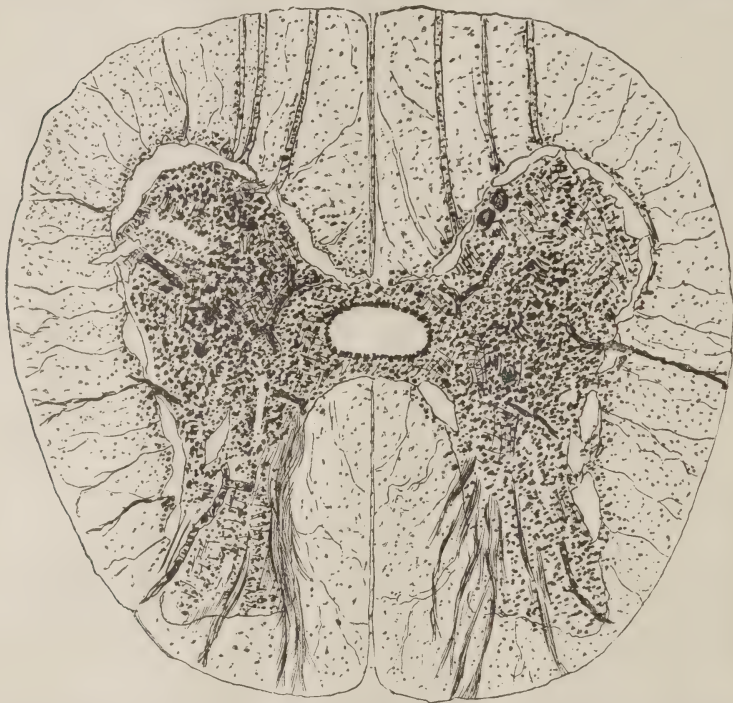


FIG. III.—Section through fifth lumbar segment of cord, twelve days after operation. All ganglion-cells of ant. horn involved. Ant. nerve-roots degenerated. Substantia gelatinosa extremely infiltrated. Post roots intact.

of the anterior nerve-roots from the central gray, remain. There is a dense accumulation of leucocytes in the substantia gelatinosa, but in other parts of the gray the increase is not much greater than before. Practically nothing remains of the anterior nerve-roots but their connective tissue sheaths, which contain small round cells, broken down

myeline, and granular débris. The posterior nerve-roots are normal.

The white substance is normal, except that here and there there is more or less nuclear increase in the proximity of the anterior horns. Along the connective tissue septa, which radiate from the gray matter, there is also some increase of nuclei. Most of these changes are shown in Fig. III.

In preparations from the cord of an animal which died on the twenty-third day, it was found that the white matter as well as the gray was distinctly diseased.

A slight amount of cellular infiltration into the white matter contiguous to the anterior horns was observed, as remarked, at an earlier period. At this time, however (twenty-third day), the process was sufficiently extensive and intense to give rise to considerable destruction of tissue in the immediate neighborhood of the anterior horns, particularly along their inner and outer borders and near the gray commissures. Many of the nerve fibres were quite destroyed, and the neuroglia in the vicinity shared in the destructive process.

Beyond this region of actual necrosis there was a zone of varying width in which the neuroglia was intact and the nerve fibres were partially or quite destroyed. The accumulations of small round cells along the connective tissue septa radiating from the anterior horns, were now more distinct than before. The total area of the gray was very much diminished, in consequence of the great destruction of tissue which at this time exists. A few necrotic ganglion cells could still be detected in the periphery, but no normal histological elements of any kind remained in the anterior horns.

The structure of the anterior roots was so obscured by cell infiltration that it was difficult to judge of their condition. It seems highly probable that their constituent elements had been to some extent destroyed or modified. There was no room for doubt about the state of the posterior nerve-roots. They were normal.

The pathological histology of the anterior horns at the end of the thirtieth day is simply an exaggeration of the condition found at this stage of the process, and the description of the former will answer for both.

Thirty days after operation the destructive process was very far advanced. The nervous elements of the central gray were all but completely destroyed. In a majority of sections obtained from the most diseased region of the cord, no ganglion cells could be detected. In some sections, however, a few cells shrivelled and devoid of processes could be distinguished. These cells were in every instance situated near the periphery of the anterior horn.

Nothing remained of the network of medullated nerve-fibres, but along the borders of the anterior horns a few degenerated fibres could be seen. No traces of the neuroglia of the gray could be detected. In consequence of these extensive destructive changes the area of the central gray was very much diminished in size, having been reduced to at least one-half its original extent. It was also very much broken up, owing to many large spaces in it, some of which communicated with the gap which always separates the anterior horn from the surrounding white substance.

The normal elements of the cord were found to have been replaced by products of inflammation and disintegration. The whole area of the gray was infiltrated with small round cells, the infiltration being very intense in the central portions of the horns. No basement substance could be detected between the cells, and it is probable that the whole process was as yet a purely destructive one.

Compound granular corpuscles were present in considerable number, and large, finely granular cells, having a diameter twice or three times as great as that of a leucocyte, were present in every part of the gray substance, or, more properly speaking, in what remained of the gray substance. Stained by Weigert's method these bodies assumed a bluish tinge, and were recognizable as the result of the breaking up and degeneration of the myeline sheaths of the medullated fibres of the gray substance. A certain amount of granular debris was also present, the origin of which is uncertain.

The anterior nerve-roots contained no nervous elements. They were infiltrated with small round cells, and in place of nerve-fibres could be seen disintegrated myeline and a few compound granular corpuscles.

The posterior nerve-roots were for the most part intact, but careful examination showed that a few of the fibres were disintegrated, possibly to some extent in consequence of pressure exerted upon them by the inflammatory products by which they were surrounded. The changes in the



FIG. IV.—Section through the fifth lumbar segment of the cord, thirty days after the operation. Total destruction of all nervous elements of the ant. cornua. Great diminution in extent of gray substance. Intense infiltration of gray with nuclei. Less intense infiltration of adjacent white substance. Striking change in contour of central canal.

white substance were of the same character as those described as having been present on the twenty-third day, but were more widely spread and rather more intense than at that time.

Fig IV. gives a general idea of the appearance of the lesion on the thirtieth day in the fifth lumbar segment of the cord. The inflammatory process affected with about equal intensity all the white tracts immediately about the

gray substance. It was impossible at the end of the thirtieth day to detect any evidence of a secondary degenerative process in the white substance, and sections from the lower end of the third lumbar segment were absolutely normal. Fig. V. represents a section of the cord from the same region, highly magnified.

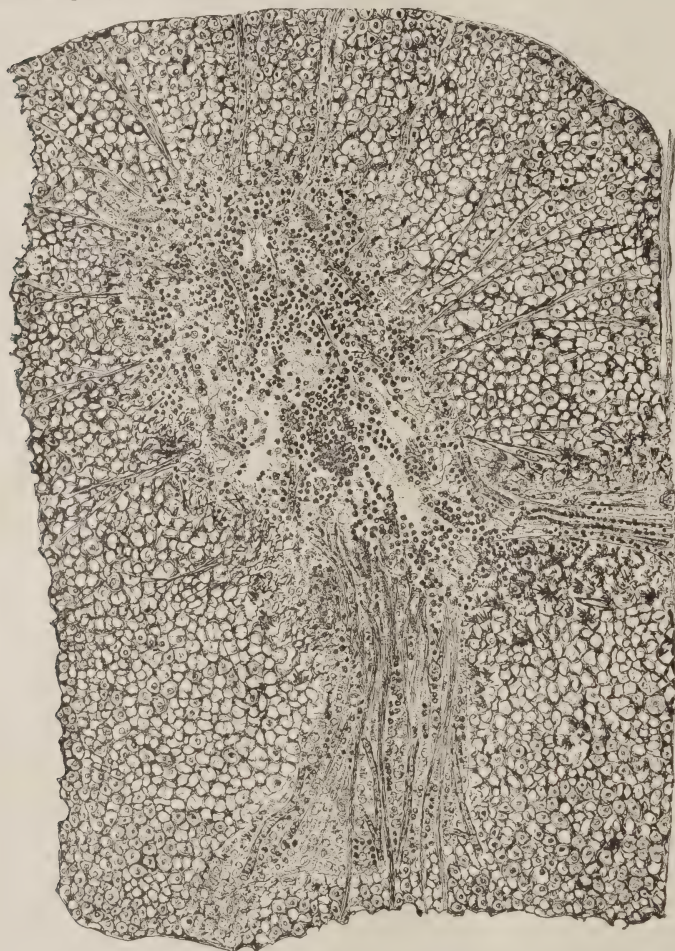


FIG. V.—Highly magnified section through the lumbar segment of the cord, thirty days after the operation.

The intervertebral ganglia of the diseased segments were the seat of minute hæmorrhages, about the middle of the first week.

Accumulations of lymphoid cells were also present by the end of the first week in the peripheral portion of the ganglion cells. This latter condition was as well marked at this period as at any subsequent time, but about the thirtieth day a new change was evident. The nerve fibres entering the ganglia from the posterior horns and those leaving it to join the anterior nerve roots, were seen to be infiltrated with lymphoid cells and to have undergone degeneration. These changes seemed less severe in the former set of fibres than in the latter. It is possible that the altered structure of the posterior nerve roots, which was evident at this time, was in some way dependent on these changes in the intervertebral ganglia.

Nerves.—With regard to the condition of the spinal nerves there is not much to be said. The changes found in the nerves corresponding to the diseased segments of the cord are for the most part such as might be expected, *a priori*, to follow the destruction of the ganglion-cells of the anterior horns. Such fibres as are derived from the anterior nerve-roots undergo a degenerative change closely resembling that which occurs in the distal portion of a divided nerve. The nuclei of the sheath are increased in size and in number. The medullary sheaths break up into segments, which subdivide and give rise to minute granules and globules, and eventually the axis-cylinder undergoes segmentation.

It seems probable that the initial changes in the nerve-fibres occur somewhat less rapidly in the cases I am describing than in the artificially divided nerves of rabbits; for I have been unable to detect complete segmentation of the axis-cylinder before the end of the third day, while in cut nerves this stage of the process is reached at the end of forty-eight hours. Subsequently the two processes appear to advance with equal rapidity.

The degenerative process affects simultaneously the whole extent of the nerve.

In two respects the histology of this condition under notice differs from the ordinary Wallerian degeneration. In the first place the proximal end of the nerve becomes the

seat of an active inflammatory process. As already stated, the anterior nerve-roots are infiltrated with leucocytes by the end of the eighth day. This process extends a variable distance down the nerve with gradually diminishing intensity, and by the end of the tenth day the blood-vessels of the nerves are found to be dilated, and the interstitial connective tissue to be extensively infiltrated with emigrated white blood-cells.

The second point of difference is that the connective tissue of the the nerves is the seat of numerous small extravasations of red blood cells, occurring during the first four or five days after the operation. These hæmorrhages are not absolutely constant. When they occur they are as apt to effect one part of the nerve as another.

Muscles.—The alterations in muscle tissue which are consecutive to the Stenson operation are confined to the muscles paralysed, and affect different muscles with different degrees of intensity. The soleus, semi-tendinosus, and adductor magnus appear to be the ones most changed.

In estimating the extent of the changes in a particular muscle, it is necessary to take into consideration whether the one in question is a red muscle or a white one, as the histology of the two kinds is somewhat different. In the red muscles, of which the semi-tendinosus may be taken as representing the type, the diameter of the individual fibres is greater than in the white ones, the number of nuclei to each fibre is greater, and there is more connective tissue between the fibres. In the red muscles nuclei are frequently found within the substance of the fibres; in the white muscles they lie beneath the sarcolemma only. Another distinguishing feature of the red muscles relates to their blood supply. They possess a very extensive capillary circulation, and on some of the capillaries one sees the dilatations which are known as capillary aneurisms. Fig. VI. represents transverse sections of normal muscle bundles, *a*, being from the abductor magnus, a white muscle; *b*, from the semi-tendinosus, a red muscle. Fig. VII. represents teased fibres from the same muscles.

About the end of the first week hæmorrhages into the

connective tissue can usually be detected. As a rule these extravasations are not extensive and do not compromise the muscular fibres. They are generally linear in form, and

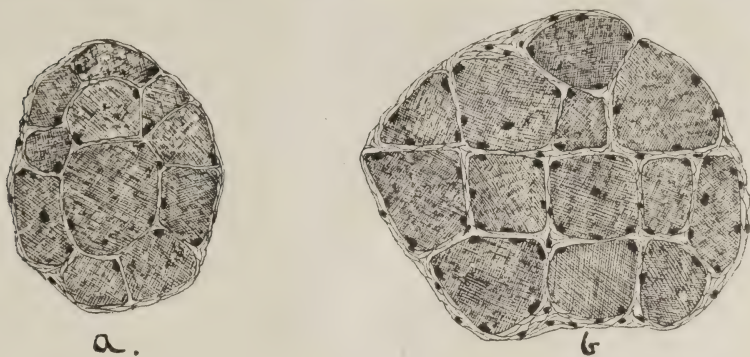


FIG. VI.—Sections through normal muscle bundles showing differences in histology of white and red muscles. (A) From adductor magnus—white muscle. (B) From semitendinosus—red muscle.



FIG. VII.—Teased fibres from normal white and red muscle, showing differences in histology. (A) From adductor magnus. (B) From semitendinosus.

their direction is that of the long axis of the muscle. Several of these extravasations may exist side by side, and thus give the appearance of a single hæmorrhage extending over a considerable area. Long rows of red blood cells derived from the muscle capillaries may separate individual fibres and may partially destroy them.

Transverse sections made through muscles at the end of the sixth day show the presence of spaces of various size and form in the protoplasm of the fibres. These spaces or vacuoles are seemingly irregular in distribution. In a particular area of a given size, for instance, the majority of the fibres are normal and only a few small vacuoles exist, while in a contiguous area of equal size the greater proportion of the fibres are affected. The presence of vacuoles in a fibre does not appear to influence the structure of the rest of the fibre; at all events, no difference can be detected by means of the ordinary staining agents.

As a rule there are two or more vacuoles in an affected fibre; sometimes there are six or seven, sometimes only one large vacuole. The longitudinal extent of the spaces varies somewhat. It is seldom greater than twice the diameter of the fibre.

In the cases I have examined, the vacuoles were most numerous about the eighth day. I have found them in large numbers as late as the tenth day, and have occasionally seen a few isolated ones as late as the thirtieth day. Both white and red muscles were affected, and with about equal intensity. Fig. VIII. shows the appearance of these vacuoles on transverse section.

A process of vacuolation, closely resembling that described, is spoken of by Schulze,⁶ of Heidelberg, as occurring in progressive muscular atrophy; and Wagner⁷ mentions the occurrence of a similar process in a case of acute polymyositis. Nothing seems to be known of the nature of the condition. I do not know what becomes of the fibres in which the vacuoles occur, nor what is the relation between

⁶ Ueber den mit Hypertrophie verbundenen Progressiven Muskelschwund, 1886.

⁷ Wagner, E. Ein Fall von acuter Polymyositis, Deutsches Archiv. f. Klin. Med. Vol. XL., 1887.

the process and the atrophic changes which in these cases follow it. There is no reason to believe that the condition is artefact.



FIG. VIII.—Section through muscle bundle, eight days after operation, showing vacuolation. From semitendinosus.

About the beginning of the third week the muscles of the lower extremities begin to waste, and microscopical examination shows the presence of a large proportion of atrophied fibres. Fig. IX., taken from a transverse section



FIG. IX.—Section through muscle bundle, twenty-three days after operation. Simple atrophy of muscle fibres. Apparent increase of nuclei. Increase of connective tissue between the fibres.

of the semi-tendinosus on the twenty-third day, shows the condition of things at this period. The majority of the fibres have undergone simple atrophy, and coincident with this change there has been a hyperplasia of the interstitial

connective tissue. Some of the fibres are very much atrophied. The number of nuclei seems to be unchanged. At all events there is no distinct loss of nuclei. Indeed there is



FIG. X.—Section through muscle bundle, thirty days after operation. From semitendinosus.



FIG. XI.—Teased fibres from semitendinosus, thirty days after operation.

an apparent increase in number, as the atrophy of the fibres has brought them closer to one another.

By the end of the thirtieth day the atrophic condition of the muscles was still more marked, the atrophy in some instances being very great indeed.

Fig. X. shows the appearance, at this stage, of the semitendinosus, one of the most atrophied muscles. Fig. XI. represents teased fibres from the same muscle. A very small proportion of the fibres were normal and some were only slightly wasted, but the great majority were greatly atrophied. The number of nuclei was so great that at first sight it appeared that they were connective tissue nuclei, and that there had been an immense increase in connective tissue. The examination of teased fibres, however, showed that the amount of connective tissue was not much greater than normal, and that the nuclei belonged to the atrophic fibres. The diameter of many of these fibres was not greater than that of the capillary. They contained a great many nuclei, but not so many as normally. In many instances a contained nucleus was wider than the fibre itself, and gave rise to a bulge at either side. The protoplasm of these greatly atrophied fibres was in a state of granular degeneration. I was unable to detect the presence of fat in them. Many of the larger fibres were also in a condition of granular degeneration, and none of them retained their striation.

The appearance of some of the smallest fibres seemed to justify the belief that they were undergoing regeneration, but on this point I cannot speak with certainty.

These changes in the muscles are undoubtedly secondary to the lesions of the cord and peripheral nerves, which have already been described.

Bladder and Uterus.—An interesting hypertrophy of the bladder occurs in rabbits which survive the Stenson operation. As early as the fourth or fifth day the bladder was distinctly increased in thickness, and by the end of the thirtieth day the hypertrophy was enormous. Fig XIII. was drawn from sections of the bladder at the latter period. There was a great hypertrophy of the muscular fibres, but probably no hyperplasia, and there was also a large increase in connective tissue. Fig. XII. represents a typical section of a normal bladder denuded of its epithelium. The con-

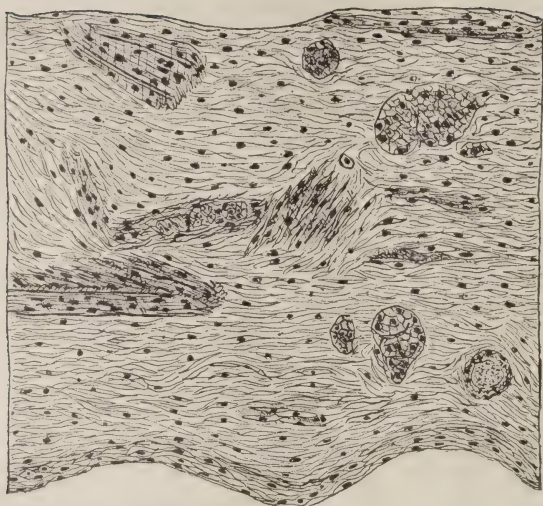


FIG. XII.—Section through normal bladder denuded of epithelium.

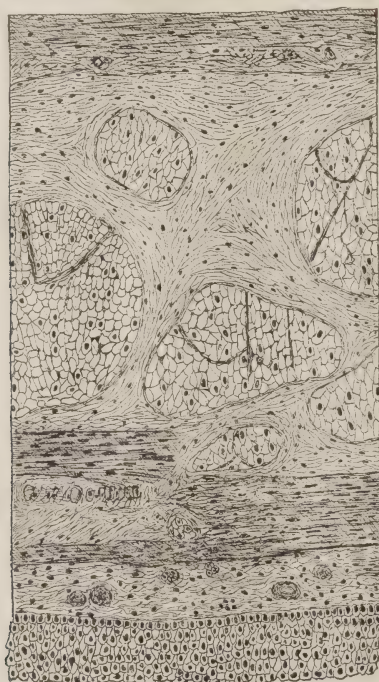


FIG. XIII.—Section from bladder, thirty days after operation. Increase in thickness of bladder. Hypertrophy of muscle-fibres. Great increase in amount of connective tissue.

trast between the normal and pathological condition is very striking. The hypertrophy is probably due to the over-distension to which the bladder is subjected, in consequence of the paralysis of the detensor urinac. The hypertrophy does not occur so rapidly when the bladder is very frequently emptied by compression from without, and it is possible that it may be altogether avoided by allowing the urine to dribble away constantly through a catheter. In sections made during the first week, hæmorrhages into the bladder wall are numerous and large.

In non-pregnant females, hæmorrhagic infarction of the mucous membrane of the uterus is one of the first consequences of the operation. In one instance, in which the uterus was examined one hour after the ligature had been applied to the abdominal vessels, several large patches of infarction, separated by areas of normal mucous membrane, were present in each half of the uterus. In another instance the infarction occupied the whole of the mucous membrane of the uterus. In this case the ligature had been on two hours. Hæmorrhagic infarction was an invariable sequel of the operation.

In Stenson's experiment we have a method of inducing a form of myelitis in animals, which in certain stages bears a close resemblance to processes which occur in man. This fact suggests that interference with the blood supply of the human spinal cord may be an etiological factor in some varieties of myelitis. A few cases have been recorded which justify the suspicion that embolism of a vessel of the spinal cord can give rise to an acute myelitis, and it is not improbable that other causes of vascular obstruction may give rise to the same effect.

The inflammatory process, which is consecutive to Stenson's operation, closely resembles, during the first two weeks, acute polio-myelitis. After this the white matter of the cord becomes more extensively involved than is ever the case in polio-myelitis.

The ganglion cells are the elements first involved, doubtless in consequence of the fact that they are the most highly differentiated structures of the cord.

Cohnheim and his pupils have shown that when the blood supply of a parenchymatous organ has been shut off, the more highly differentiated elements are the ones which suffer first; and in 1869 Schiffer^s called attention to the fact that when the blood supply of the spinal cord has been cut off, the white substance and nerves retain their excitability long after the gray substance has reacted to the anæmia.

It is probable that the large number of small round cells with which the gray matter is infiltrated eventually becomes organized into connective tissue. If this is really the case, the region of the cord affected by the inflammation must in the course of time become transformed into a slender fibrous cord.

It occasionally happens that instead of destroying a large number of ganglion cells, the inflammatory process involves only a few groups of cells. These are the cases in which only a few of the muscles of an extremity are paralyzed. A careful study of the limits of the lesions in these instances would doubtless throw light on the functions of the ganglion cells of certain levels of the spinal cord.

It was my object to secure the greatest possible uniformity in all the essential conditions of the experiments upon which the foregoing observations are founded. In all instances the time during which the vessels were compressed and the level at which the compression was effected were approximately the same. By carefully varying these conditions, one at a time, interesting facts relating to the anatomy of the spinal cord would doubtless be brought to light.

It is proper that I should give a few directions as to the manner of doing Stenson's operation, for even when the greatest precaution is observed in performing it, the number of animals that die within the first three days is great. Without the precautions which constitute the technique of the operation the mortality is so great as to be discouraging.

After the third day deaths referable directly to the operation are uncommon, but as it is a matter of consider-

^s Centralblatt f. die med. Wissenschaft, 1869, No. 38.

able difficulty to keep the paraplegic animals alive after the third week, in consequence of the failure of general nutrition, it is better to have a number of animals in progress at once than to centre one's hopes of long-lived cases in one or two rabbits.

Rabbits slightly above the medium size seem to bear the operation better than those which are either very large or very small. Female rabbits are not eligible, because death generally occurs very early, in consequence of the infarction of the uterine mucous membrane already spoken of.

It is of great importance that the general condition should be good, and that the thermometer should not stand above 85° in the shade at the time of the operation.

The animal should be fastened with the belly down on a suitable holder. A holder for the head is only necessary when large rabbits are used.

The hair is now cut from a rectangular area of the back, the upper and lower limits of the rectangle being respectively the spinous processes of the first lumbar and first sacral vertebræ, the lateral border being two or three inches distant from the median line of the back. The skin should next be sterilized by cauterizing it superficially over an area of about one inch in diameter on each side of the vertebral column. The centres of these areas should correspond to the points at which the needle enters and leaves the abdominal wall. These points are symmetrical, and are located by the intersection of lines corresponding to the outer border of the quadratus lumborum of each side, with a line drawn at right angles to the vertebral column at the level of the fourth lumbar spine.

All the skin denuded of hair is next sterilized with a solution of bichloride of mercury (1-1000), and in all the manipulation that follows every care should be taken to prevent micro-organic infection.

The needle employed in the operation is represented in Fig. 14. It consists of a slender curved trocar with a large eye at one end and a fine point at the other. The needle is sheathed nearly, but not quite, throughout its length

in the curved canula, *a, a*, which is provided with a guard, *b*. The curvature of the needle should be that of the arc of a circle, the sector of which arc subtends an angle of about 170 degrees. A needle in which the radius of curvature is about one and one-third inch will be found more generally useful than any other.



FIG. XIV.—Needle used in doing Stenson's operation. (*AA*) Canula. (*B*) Guard. (*C*) Eye of needle.

Before passing the needle, a very small incision should be made through the skin and subcutaneous tissue, near the centre of one of the cauterized areas mentioned. The point of the needle is pushed gently through the abdominal wall and then drawn back into the canula, which is next pushed cautiously through the abdominal wall and peritoneal cavity until it reaches the peritoneum lying below the cauterized space of skin of the opposite side. The point of the needle is then protruded from the canula and the abdominal wall is pierced from within outward, the needle being entirely withdrawn through the new opening. The canula is taken out through the opening first made. The ligature carried by the needle in its passage through the animal should be of silk, in preference to catgut, silkworm gut or silver, and should not be so fine as to injure the tissues included by it, nor so coarse as to render effectual

compression of the vessels difficult. I have found that a ligature of four strands or ordinary knitting silk answers the requirements.

The degree of tension required to compress the vessels sufficiently to check the blood flow through them varies considerably in different instances, but is never very great if the operation has been properly performed. In order to be able to regulate the tension upon the ligature, I have found it very convenient to employ a small saddle-like

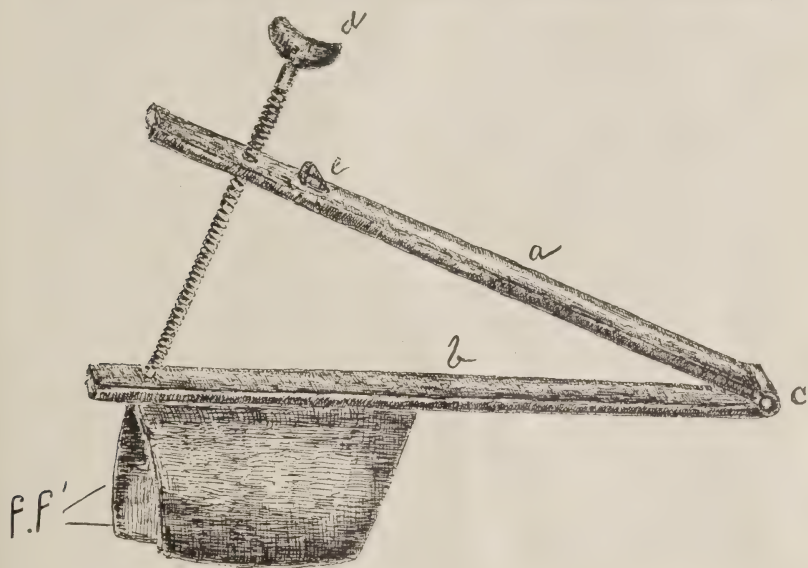


FIG. XV.—Tension saddle. (A) Upper piece. (B) Lower piece. (C) Hinge. (D) Screw. (E) Nose. (F F') Copper plates.

instrument, which is placed over the lumbar vertebræ, and over which the ligature is tied. This "tension saddle" is shown in Fig. 15. It consists of two thin pieces of iron, a, b, about three inches in length and one-half inch in breadth, joined by a hinge, c. The posterior end of the upper piece, a, is perforated by the screw, d, and on either side of the lower piece, b, is fastened a triangular plate of copper, f, f'. These pieces serve to steady the instrument when in position, and may be adjusted to fit the back of any animal. When the saddle is in use the screw is with-

drawn sufficiently to permit the pieces, a, a, to lie in contact. The ligature is then tied tightly over the nose, e, on the upper piece, and the desired tension is obtained by separating the parts, a, b, by the screw.

Accidents may occur which either prove fatal to the animal or prevent the success of the operation. Of these the following are the most common: Inclusion of a loop of intestine in the the ligature; injury of the kidney; puncture of the intestine and escape of contents, and puncture of the aorta or vena cava. The shock of the operation often suffices to kill the animal. Anæsthetics are always dangerous and should never be used. It is possible to pass the needle between the vessels and the bodies of the vertebræ. This happened in two cases, in which it was, of course, impossible to compress the vessels. Peritonitis may occur from injury to the intestine or from infection from without.

Suppuration in the muscles and structures in the path of the needle is frequent if aseptic precautions are not used.

Pregnant females often die in consequences of separation of the placentæ from the uterus, the result of hæmorrhage. In non-pregnant females fatal infarctions of the uterine mucous membrane are a common sequel of the operation.

The ureters are invariably included in the ligature, but this inclusion seems to do no harm.

The foregoing pages were written in August, 1887, but publication was delayed.

On November 10, 1887, a paper was read by Singer before the Wiener Akademie on the subject of experimental myelitis. Singer's results were in the main confirmatory of those of Ehrlich and Brieger.

In January, 1888, a much better article by Spronck of Utrecht appeared in Brown-Sequard and Charcot's journal. Spronck describes with especial detail the anæmic necrosis of the ganglion-cells, and finds that distinct changes appear in the cord as early as twenty-four hours after the operation (Stenson's). He is very positive that the posterior nerve-roots undergo no change, but I believe the degenera-

tion of the posterior roots in some of my specimens to be unquestionable. The abundant nuclei in the gray substance to which I have referred in my paper as emigrated white blood cells are regarded by Spronck as the result of proliferation of the neuroglia nuclei.

This opinion is based on the fact that karyokinetic figures can be demonstrated in some of these nuclei. It is indeed more in harmony with the doctrine that the fixed cells, especially connective tissue cells, are incited to increased production by the falling out of the parenchymatous elements (in this instance necrotic ganglion-cells) that the new nuclei should belong to neuroglia rather than leucocytes. Julius Arnold, however, has shown in a recent article in the *Archiv. f. Microscopische Anatomie* (Bd. xxx., H. 2, S. 205), that migratory cells probably proliferate by caryokinesis as well as by direct nuclear fission, and Kultschizky (*Medic. Centralblatt*, No. 6, S. 97-98,) is positive that the white blood cells multiply by karyokinesis; Kultschizky even goes so far as to state that direct nuclear fission never occurs.

After consideration of these facts it becomes evident that Spronck's view concerning the nature of the accumulated nuclei is certainly open to question, and I think we may fairly say that there is about as much evidence that the cells are leucocytes as that they are neuroglia cells; or, to put it more accurately, that there is as little direct evidence in one case as in the other.

A CASE OF TUMOR OF THE CERVICAL REGION OF THE SPINE.—OPERATION AND DEATH.¹

By JAMES HENDRIE LLOYD, M.D.,

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AND

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MEDICAL REPORT BY DR. LLOYD.

THIS patient was admitted into the writer's wards at the Philadelphia Hospital as a simple case of hemiplegia of several months' duration, and as such cases abound there, and as she did not present obtrusively any special symptoms, several days elapsed before she was prominently brought to my notice. The resident physician, Dr. Talley, then called my attention to the fact that the patient had stiffness of the neck, pain on moving the parts, and a slight prominence on the left side of the spine at about the level of the third cervical vertebra. The following history was elicited:

Catharine W., aged about forty-five years, native of Germany. No history of tubercle, alcoholism, or syphilis obtainable. Four months before her admission to the hospital she said that she had a burning pain in her head. About this time the left leg became weak and she gradually lost power in it. The left arm then began to lose power. It was not stated by the patient very accurately how long after the leg the arm became paretic, but it was a

¹ Read before the American Neurological Association, at the meeting of the Congress of American Physicians and Surgeons, Washington, D. C., September, 1888.

period of some weeks. As in the case of the leg, the paresis of the arm was gradual in its approach until the limb was quite paralyzed. There was not, and never had been, any paresis of the face or tongue. For some time (period not accurately stated) before the appearance of any paretic symptoms the patient had noticed a slight swelling or tumor on the back of the neck immediately to the left of the median line, and corresponding to the third cervical vertebra. The neck was stiff. During sleep she said her arm "jerked."

Her condition at the time of the first examination was as follows: The left arm was paretic and slightly wasted (?). The left leg was paralyzed, slightly spastic, and gave well-marked ankle clonus and rectus clonus. The patellar reflexes were very much exaggerated in both legs. The muscles of the affected limbs gave no changes to electric excitation. The right arm and leg were positively normal in their motor functions to the most rigid tests, but there was slight ankle clonus on that side, not nearly so great as on the affected, or left, side. There was no anæsthesia anywhere. Mindful that this was a case in which crossed anæsthesia and paresis might exist, I examined the patient's right (sound) arm and leg with the greatest care again and again, but no tests demonstrated any loss of sensation. The same was true of the left (affected) side. There were no subjective symptoms of altered sensibility (paræsthesia), nor to heat or pain. There was no paralysis of any facial, ocular, or lingual muscles of either side, nor any alteration of sensation in these regions. The pupillary reflexes to light and accommodation were normal. She had a marked swelling on the back of the neck, referred to above. It was slightly sensitive to pressure, and seemed very deep seated. She was very positive in her statement that the tumor in the neck came on four months before the paralytic symptoms, and that these paralytic symptoms came on gradually, beginning in the leg, and afterward involving the arm. She had had intense pain in the cervical region. Dr. de Schweinitz examined the eye-grounds, and found evidence of a slight retinitis in each eye. There were no vasomotor or trophic disturbances.

In consultation with Dr. John B. Deaver the case was again carefully examined, and the tumor was considered to be probably a growth from the inside of the spinal canal, extending out and slightly compressing the cord. As the cord-compression was yet very limited in area, only involving the left lateral tract, while threatening every day to invade new territory with fatal results, and as the woman was already badly disabled, it was decided that an exploratory incision should be made. This opinion was endorsed at a subsequent consultation by Dr. D. Hayes Agnew, who kindly saw the case. The operation was accordingly performed July 17, 1888, by Dr. Deaver, in the presence of Drs. Agnew, Ashhurst, and Mills.

The details of the operation are given by Dr. Deaver in his paper. The laminæ of the third and fourth cervical vertebræ were cut through and the spinous processes removed, thus exposing the theca. The swelling on the side of the spine was found to be thickened and somewhat displaced bony tissue of the vertebra. The bones were evidently the seat of an inflammatory process, and were softer than normal.

The dura mater was thickened and opaque. There was no bulging or swelling within it. As there was no indication for more interference, it was decided not to open the membranes, and accordingly the operation proceeded no further. To all appearances, the whole of the pathological process had been confined to the bones and to the soft parts without. Evidence was altogether lacking in the operation itself of what the exact condition had been which caused such circumscribed pressure upon the cord as only to impinge upon the left motor tract. The respiration of the patient was altered during the latter part of the operation, and again before her death three days later. This alteration consisted in deep, almost gasping inspirations with quite a prolonged interval between. Whether this was caused by any interference with the functions of the phrenic nerve, which is described by some as originating in the fourth cervical segment, I am not able to say. It ceased as the patient rallied from the ether, but returned again a few hours before her death.

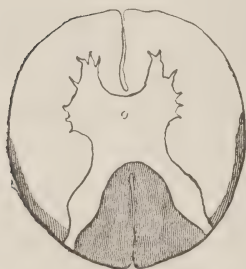
The patient's paralysis, after the operation did not improve. Her arm and leg were tested as carefully as her condition would admit; she was very weak, but evidently made an unsuccessful effort to throw the palsied limbs into motion when requested to do so. On the third day after the operation her condition changed for the worse, the respiration was altered as described, she became unconscious and died. It would be difficult to explain exactly the mechanism (so to speak) of her death. It was not expected up to a short time before her end. The surgical conditions were aseptic and good. There was no increase of pressure upon the cord; at least there were no new paralytic symptoms to indicate such. The wound had ample drainage. The alteration in the respiration alone seemed to furnish an explanation.

The notes of the *autopsy* are as follows: The cord presented a perfectly normal appearance up to the region of the operation, where the dura was slightly thickened and congested. On splitting up the dura there was observed on the anterior aspect of the cord at about the level of the second cervical nerves a slight prominence on the left side about five-eighths of an inch below the decussation of the pyramidal tracts. This prominence was of the same color and apparent consistence as the cord, was somewhat conical, and about the size of the end of a wheat grain. The cord was not cut, but preserved for microscopic examination. Careful examination of the external and internal capsules of both the right and left hemispheres and of the motor tracts through the crura and pons revealed a perfectly normal condition. At the base of the brain the membranes were not involved, and the basilar and other arteries, including the arteria hæmorrhagica and middle cerebral, were not atheromatous. The ventricles contained a normal amount of fluid without blood. The centrum ovale was normal, as was the motor cortex to naked eye inspection. The kidneys showed a slight tendency to congestion and to interstitial change.

I am indebted to Dr. E. O. Shakespeare for the following report on the pathological appearances of the sections of the cord which he made:

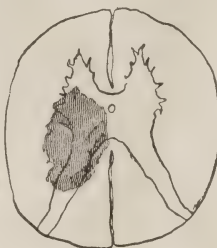
Three segments of the cord were examined—the one above the lesion, the one containing the lesion, and the next one below. The top of the upper segment shows oil globules in the posterior columns and in the cerebellar tracts. Below in this segment is a circumscribed lesion involving the gray matter from the middle line forward as far

FIG. 1.



Three-quarters inch above projection, showing area of ascending degeneration.

FIG. 2.



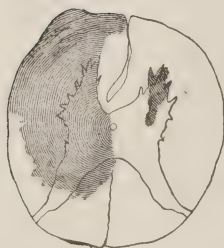
Above projection, showing area of hæmorrhagic extravasation.

FIG. 3.



Still above projection, showing area of hæmorrhagic extravasation.

FIG. 4.



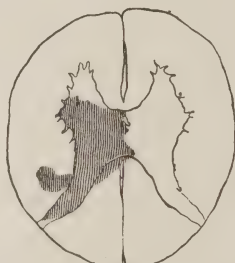
Top of projection, showing projection and area of hæmorrhagic extravasation.

FIG. 5.



Little below projection, showing area of hæmorrhagic extravasation.

FIG. 6.



Bottom of middle segment, showing area of hæmorrhagic extravasation.

as the beginning of the anterior horn and back to one-half the length of the posterior horn; it also involves the crossed pyramidal tracts; also slightly involves the multipolar cells in the anterior horn. An upper section from the middle segment involves the gray matter beginning about at the posterior edge of the central canal back to the edge of the

gray commissure, and involving the posterior left horn and the outer border of the gray matter extending into the anterior horn of the left side; the whole of the left lateral column is involved at this level, including, of course, the motor tracts. The posterior root zone is here also slightly involved. There is a small area of involvement on the right side in the anterior column; also slight at exit of the right anterior nerves. The middle sections of the middle segment show unilateral involvement of the gray matter (left side) and limited involvement of the trophic cells on the right side. The anterior part of the columns of Burdach is involved. The whole of the lateral column is involved (this section corresponding to the level of the projection seen post-mortem), and on the anterior part is an intensification of the lesion. The lower section of the middle segment involves about the same as the last, except that the column of Burdach is involved further back. The right anterior horn is slightly involved, also a limited islet in the right column of Burdach near the column of Clark. Middle of lower segment shows the lesions limited sharply to the gray matter of the left side posterior to the central canal, anterior parts of the posterior horns, and the crossed pyramidal tracts. The lesion consists, in the main, of an extravasation of blood with a more or less complete destruction of the gray and white matter involved. There is also some inflammatory action, principally in the neighborhood of the peripheral portions of the hæmorrhage.

OBSERVATIONS.—The present appears to be an era of operative interference in diseases of the cerebro-spinal system. It is well, therefore, to publish both the unsuccessful and successful cases. From the former is sometimes learned more than from the latter. This case, it now appears, was beyond the aid of surgery. The lesion was too far anterior, and too deep in the substance of the cord itself to be reached by the knife. It was, at least, an instance of successful localization of a lesion in the cord. The manner of the patient's death creates a suspicion that the phrenic was in some way interfered with; and this may serve as a

possible danger-signal to those who are about to operate upon the cervical spine. On the other hand, it must be remembered that in our case the bones were involved in an inflammatory process.

SURGICAL REPORT BY DR. DEAVER.

July 16, 1888.—The day previous to the operation the patient's bowels were moved with a saline purgative; the urine examined chemically and microscopically, proving to be negative; and the heart and lungs carefully examined, eliciting no organic trouble. She was given first a warm water bath, then a boric acid bath, after which the neck was washed with turpentine, then scrubbed with soap and water, again washed with ether, alcohol, and enveloped with a towel wrung out of a solution of 1 : 1000 bichloride of mercury.

Operation, 17th, 1 P. M.—The patient having been etherized, was placed upon the table, and with Prof. D. Hayes Agnew and Prof. John Ashhurst, Jr., assisting, and in the presence of Drs. J. Hendrie Lloyd and Charles K. Mills, I made a longitudinal incision over the median line of the nape of the neck, extending a little distance above and below and down to the spines of the affected vertebræ; then separated the muscular attachment and reflected the soft parts laterally on either side as far as the junction of the transverse process with the pedicles of the vertebræ, thus clearing the spinous processes, as well as the laminæ of the third and fourth cervical vertebræ and their ligamentous attachments, when was exposed a bony tumor, convex from above downward and from side to side, being much greater upon the left side. The soft parts thus far dealt with were, macroscopically at least, normal.

The spinous processes were removed at their base with a pair of bone pliers, then the laminæ on each side were divided behind the articular processes with the same instrument. Upon making the section of the spinous processes, they were found to be a trifle softened, while the laminæ, particularly on the left side, the side corresponding to the

most prominent part of the tumor, were softened and enlarged, the cancellous tissue of same containing some pus; in other words, the condition was that of chronic osteitis with perhaps some osteo-myelitis. Upon the removal of the laminae, which were adherent to the dura mater, the dura mater was seen intact at the bottom of the wound, the connective tissue normally existing here having been absorbed; neither were there any bloodvessels present (the posterior longitudinal spinal veins and their connecting branches, which exist here normally, being absent). The dura mater did not rise up into the bottom of the wound when the bone was removed; it presented an opaque appearance and was quite resisting to the sense of touch, and appeared thickened, having shared in the inflammation of the bone. It was opened with an exploring needle, with purely negative results, and was tougher than normal.

The operation, so far as removing any more tissue, was completed, and it remained to readjust, fix, and dress the soft parts.

A rubber drainage-tube (medium size) was placed in the wound, the muscles and the deep fascia covering them sewed with catgut, and the skin and superficial fascia with silver wire. The wound was dressed antiseptically, first dusting on iodoform, covering with protective, wet with a solution of 1 : 2000 of bichloride of mercury, twelve layers of bichloride gauze, wrung out of a solution of 1 : 2000 of bichloride of mercury, twelve layers of dry bichloride gauze, these covered with bichloride cotton, and lastly, with an antiseptic bandage. During the operation the strictest antisepsis was observed, the wound being continually irrigated with a solution of 1 : 4000 of bichloride of mercury. Immediately after the operation the temperature and pulse were normal, the respiration being of the character described by Dr. Lloyd.

Patient sent back to ward; ordered application of dry heat to body and extremities; ammonium carbonate, grains five every two hours; milk and lime-water.

4 P. M., after the operation, the temperature was 97° , respiration 21, pulse 104. 6.30 P. M., temperature $96\frac{2}{3}^{\circ}$, pulse 92, respiration 28.

18th.—Patient passed quite a comfortable night and seemed as well as before operation; mind perfectly clear; pulse 80, respiration 28, temperature $99\frac{2}{5}^{\circ}$; dressings examined and found to be dry and in position. The respiration being of the same character as spoken of above, I did not think it injudicious to order $\frac{1}{150}$ of a grain of sulphate of atropia hypodermatically twice daily.

19th.—The patient's breathing becoming more laborious, and her general condition alarming the resident physician, I was summoned to go to the hospital. Upon my arrival, at 12 M., the condition was as follows: pulse 100, respiration 32, temperature $99\frac{4}{5}^{\circ}$. The respirations being quite shallow, electricity was applied; one pole over the side of neck corresponding to the origin of the phrenic nerve; the other, to the lower margin of the chest, with the idea of stimulating the diaphragm. I ordered the ammonium carbonate and the atropia to be continued, with the addition of one-half ounce of whiskey every two hours. The dressing being slightly soiled, I dressed the wound, when I found it healed except the tract containing the drainage tube; there being no discharge from this, I removed it and washed out the tract with a solution of 1:2000 bichloride of mercury.

20th.—No improvement; respiration 40, pulse 150, temperature $100\frac{3}{5}^{\circ}$. Thinking that her condition might be partly due to pressure from collection in the bottom of the wound, I removed the dressings, took out two stitches, broke up the union and, examining the wound very carefully, I found it to be clear; I therefore dressed it and ordered the treatment continued.

Patient died the following morning at 4.30.

The cause of death I attributed to inhibition of the phrenic centre, there being no other possible explanation at which I could arrive.

My reasons for attributing the cause of death to phrenic inhibition are that, prior to the operation, the respirations were normal, and that during the early part of the operation nothing abnormal was noted in the breathing, but at the

latter part it was noticed to be changed and answering to the description given by Dr. Lloyd. The only explanation I can give for this is that the spinal cord about the position of the phrenic centre might possibly have been injured with the exploring needle, or by some other condition not recognized, the operation, up to the time of introducing the needle, could not in any way have injured the cord, as the dura mater was in no way interfered with, with the exception of separating it from the adherent laminæ when it was left intact at the bottom of the wound, and the needle passed through it to determine whether or not there was any further mischief. In the future, if I have the opportunity to operate upon the cervical portion of the spinal cord, I certainly will not use the exploring needle as a means of diagnosis, but, in preference, will lay open the dura mater and expose to inspection the deeper parts; this being, to my mind, a cleaner, more satisfactory, and, to the patient, a less dangerous procedure. In operations upon the cord in its remaining regions, the use of the exploring needle would not be so objectionable. I believe this is the first time in this country that an operation has been undertaken with the view of removing a spinal cord tumor, and that but two other surgeons elsewhere have preceded me, namely, Mr. Macewen and Mr. Horsley. Mr. Macewen's cases were not really tumors of the cord, but of the connective tissue between the dura mater and the spinal canal. In view of my again meeting with a similar case, I certainly would advise operative interference carried to the extent of exploration at least, which shall determine the advisability of proceeding further, and I do not consider that the result here obtained should discourage us at all in furthering the good work already done upon the cerebro-spinal axis.

FOOT NOTE.—We are indebted to the *Journal of the Medical Sciences* for permission to publish this article.

THE THERMO-POLYPNŒIC CENTRE AND THERMOTAXIS.¹

BY ISAAC OTT, M.D.

IN a paper recently published by Prof. Richet on what he calls a new function of the medulla oblongata, he states that in dogs exposed to a temperature of 86° F., with the elevation of the body heat of the animal the number of the respirations suddenly increased 350-400 per minute, a form of respiration which the author calls polypnœa. He shows by numerous experiments that it is not the want of oxygen which causes polypnœa, for it is necessary that the animal be not in need of breathing, but in a state of apnœa, for polypnœa to occur. An excess of carbon dioxide in the blood interferes with polypnœa. An animal pants to cool himself, whilst a man perspires under the same conditions. The role of polypnœa is to regulate the temperature of the body exclusively, as was seen in the experiment upon two dogs exposed to an equally high temperature, one of which was curarized or otherwise so manipulated as to interfere with polypnœa; the temperature of the curarized dog ran to 110° F., while that of the other did not go higher than 103° F. This new function of the nervous system Richet calls thermo-polypnœa—a reflex function, he states, ordinarily, but when it is insufficient central it regulates temperature by an exhalation of vapor from the skin or from the lungs. Section of both vagi did not alter the course of the phenomena. The application of heat to the bodies of animals has been studied by many observers. Ackermann² arrived at the conclusion that not only the skin of animals but also the lungs are used as an apparatus to regulate the body-temperature, the skin acting in a more gross manner, the respiratory mechanism being used in bringing about the minor variations. He also states that

¹ Read before the New York Neurological Society, January meeting.

the frequency of the respirations is caused neither by the want of oxygen nor excess of carbonic acid in the blood, but alone in the increase of the temperature of the whole organism, a heat dyspnœa. When the temperature of the animal is high, artificial respirations have no effect on the frequency of the respirations, not even when, in consequence of the inflations, the venous blood is bright red; while at a lower temperature artificial respirations can greatly reduce or even bring to a standstill the movements of the respiratory apparatus. Dr. Goldstein,³ who also has greatly extended the experiments upon this subject in Fick's laboratory, asks, "Is the increased temperature of the blood a new cause adding itself to the stimulus for the respiratory centre normally present in the blood, or is it an influence diminishing the resistance." He also inquires if it acts on the cerebral convolutions primarily and through these on the respiratory centres, or does it act on the skin first, or on the pulmonary endings of the vague first, or, finally, does it act directly on the nerve centres which govern the respiratory movements. He arrived at the conclusion that the heat acted on the blood, elevating its temperature, and the heated blood acted directly on the respiratory centre, causing the increase of respiratory movements.

Dr. Sihler,⁴ at Johns Hopkins University has also made a number of experiments upon this subject, and arrived at the conclusion that the animal cannot be made apnœic, that cutting the pneumogastrics does not prevent the increase in the respiratory rhythm, nor does opium, although Richet found chloral to do so.

He does not fully agree with Goldstein that the cause is in the blood acting directly upon the respiratory centres, but holds that the increased respiration following exposure of the animal is due to two causes, skin stimulation and warmed blood; of these, skin stimulation is the more powerful. That apnœa can be produced if the spinal cord is cut,

² Deutsche Archiv. f. klinische Medicin, 1867

³ Arbeiten aus den physiologischen Laboratorium der Würzburger Hochschule, 1872.

⁴ Journal of Physiology, vol. ii., No. 3.

thus removing greatly the skin stimuli, and that the direct action on the respiratory centres of the hotter blood of the heated animal is probably not, or not only, due to its temperature but to its greater venosity. Gad and Mertschinsky have also investigated the subject, and they believe that increased temperature of the blood stimulates the respiratory centres or increases their excitability.

The latest writer upon hyperthermia due to external heat is Vincent.⁵ He proves what has been surmised before, that the blood of an animal dying of excess of heat contains a poison which causes convulsions, stupor and death in guinea pigs, sparrows and frogs. Comparative experiments with normal blood on the same kind of animals were without effect.

The experiments upon which this paper is founded were made upon rabbits and cats. They were etherized, bound down, their skulls trephined, and the cortex broken up with a blunt probe, so as to prevent any perception of pain during the whole of the experiment. With the polygraph of Marey was connected a T canula, which was bound in the trachea. Through this arrangement the number of respirations were written on the smoked drum of a kymographion of Ludwig. To study the effect of heat on the normal animal, a sheet-iron box lined with wood and large enough to accommodate Czermak's holder, upon which the rabbit lay, was heated by a Busen burner beneath. A thin layer of water was kept on the bottom of the box to prevent the wood from being charred. The top of the box was partly closed by a woolen covering, although not enough to prevent a full interchange of air in the box. The temperature of the box was usually 100° F., but at times was higher and lower. A thermometer hanging in the box noted its temperature. After the normal respiratory movement of the animal was noted, he was placed in the box about five minutes, when the curve again was taken and the rectal temperature noted. The observations were taken every five, ten, or fifteen minutes, according to the circumstances which I wished to study.

⁵ Recherches experimentales sur l'Hyperthermie. Par le Dr. H. Vincent, 1887.

When the cortex alone was removed, heat still caused a great increase of respiration. If, however, the corpus striatum and the parts between it and the optic thalami were removed, then the respirations at first began to fall instead of rising, but at high rectal temperature rose to a little above normal. If, however, a puncture was made by a blunt "seeker" into the tissues between the corpora striata and the optic thalami, then no increase of respiration ensued, except a small amount on high elevation of rectal temperature just before the respirations began to fall. In Fig. 1 are represented the curves of temperature and respiration with cortex removed. In Fig. 2 are shown the curves of temperature and respiration when corpora striata and parts between them and optic thalami are removed. In the first figure the curve of temperature and respiration ascend together, till the rectal temperature reaches a high point, when the respiration curve begins to descend and continues to do so till death ensues, this lethal temperature point being about 111° F., a fact shown by several observers. In the second figure the temperature curve rises as in Fig. 1, but more abruptly, whilst the respiration curve begins to descend, and only at limits of temperature approaching the death-point does it rise to normal or a little above it. If the animal is breathing rapidly, and a probe be thrust into the tissues of the brain between the corpora striata and optic thalami, then the respiration is reduced greatly, and remains so, no matter what the internal temperature of the animal. If, with a pair of electrodes insulated to near their point with sealing-wax and attached by a wire to an upright on the rabbit-holder to steady them, an induction current is sent through them for about three minutes, then the respirations in the animal with his cortex removed are doubled or trebled in number.

In Fig. 3 the line marked 1 is normal, and during the 2d, 3d, and 4th curves the current is acting on the centre to produce polypnœa, which is shown in the curve marked 5. The current was so weak as scarcely to be perceptible to the tongue, and the electrodes were pushed to a point lying just over the parts between the corpora striata and

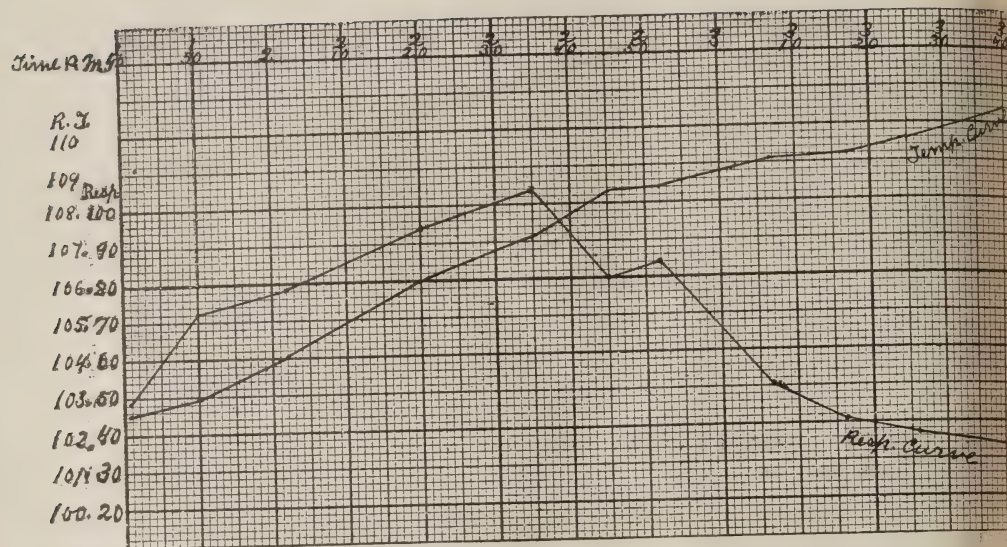


Fig. 1.

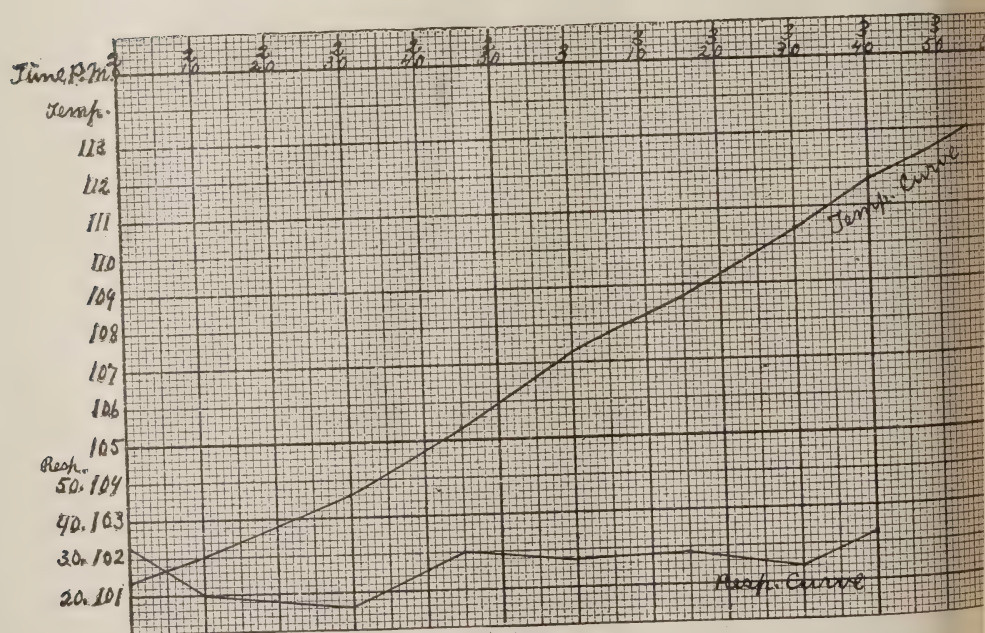


Fig. 2.

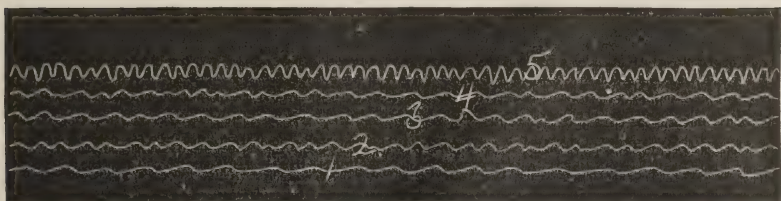


Fig. 3.

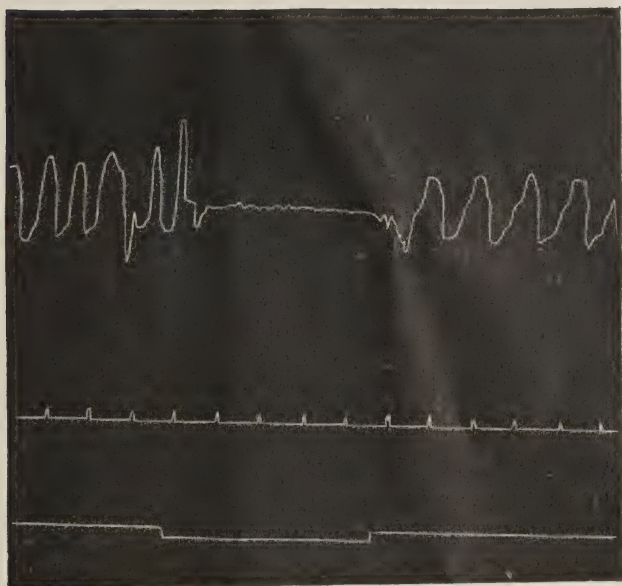


Fig. 4.

optic thalami. In some experiments a single induction shock per second was sent through the centre above mentioned, and arrest of the chest in expiration obtained. This is seen in Fig. 4, which is to be read from right to left.

To establish a centre, three things are necessary: 1st, that its abolition causes the phenomena to disappear; 2d, that irritation—mechanical, chemical, or electrical—causes the phenomena to be present; 3d, that the part of the nervous system exhibiting these peculiarities is circumscribed in extent. If now I apply these tests to the polypnœic centre, it is found that the function disappears when this point is destroyed, and that it appears when the same part is electrically irritated, and, third, that this centre or point is circumscribed in extent. It seems to me that the polypnœic centre is fully established. This discovery also shows that in lower animals external heat acts in a reflex manner upon the polypnœic centre, and not directly upon the respiratory centre, as has been held by Richet and others; that the sensory nerves send impulses into the polypnœic centre, which acting as a reflex centre, sends other impulses which are carried to the respiratory centre in the medulla oblongata and causes rapid respiration. These facts lead us to think that fever in man excites rapid breathing in a similar manner; that heat is the best external stimulant to rouse, through the polypnœic centres, the centre of respiration in cases of poisoning or drowning. I have twice seen the best effects in cases of opium poisoning, where the respirations were about six per minute, by the immersion of the feet into water of a high temperature.

If we examine the respiratory apparatus, it is found that the nervous mechanism is as follows: 1st, a main centre in the medulla oblongata; 2d, Christiani's expiration centre; and, 3d, Martin and Booker's inspiration centre in the posterior bodies of the corpora quadrigemina; 4th, Christiani's inspiration centre in the thalami at the anterior edge of the corpora quadrigemina. It occupies the centre of the thalami exactly in their median line, and, if I may be permitted, a fifth centre, the polypnœic. It might be observed that both Christiani and Martin have established their cen-

tres solely by irritations; no effect of extirpation of these points has been shown. I mention this fact to show that observers capable of forming a judgment believe the fact of irritation sufficient to establish centres, not that I doubt their experiments or their conclusions.

Christiani states that the optic, auditory, sensory nerves of the skin, and certain fibres of the vagus, stand in relation with the thalamic inspiration centre. The inhibitory nerves, according to him, in connection with these centres are the other fibres of the vagus not mentioned above, the nerves conveying painful sensations, and especially the trigeminus.

Having shown that a polypnœic centre exists, it remains to determine why after its removal the number of respirations fall from a high temperature. When I cut the trigemini within the skull, they still fell; when I removed the thalami they also fell; when I removed the corpora quadrigemina, there was also a decrease of the number; the same ensued when only the pons and medulla oblongata were left. If, however, the pneumogastriks were divided, then hardly any fall ensued. I noted, in a few experiments where only the pons and medulla were present, that with the vagi intact a temporary rapidity of respiration soon followed by a fall. In one case, where the dividing line between the pons and medulla was nearly reached, no fall ensued, and the increase at the start lasted. This increase, however, was not over about ten or eleven in number of respiration per quarter minute.

In the Weigert treatment of phthisis, by air heated from 212° – 482° F. and inhaled, the respirations are found to be diminished, although the temperature of the body rises one to two degrees for an hour and then subsides.

Here the superheated air calls into activity the inhibitory fibres of the vagi, thus diminishing the activity of the respiratory centre. In irritations of the polypnœic centre by the electric current, I have frequently seen convulsions ensue, although the cortex had been removed. When a rabbit is heated up and polypnœa occurs, plunging the rabbit into ice-water brings the respiration to normal, although

the respirations are very deep. But this return to normal lasts only fifteen seconds, when the respirations again ascend nearly to their original rapidity and then gradually descend. If ten grains of antipyrin are given subcutaneously, and the animal heated, polypnœe does not take place; but if polypnœa is first thoroughly established, and eight grains of antipyrin are injected by the jugular, it takes about fifteen minutes for the breathing to return to normal. Injection of putrid blood, causing septic fever, seemed to partially prevent polypnœa; for, when the animal was heated up, the respirations did not attain their maximum frequency. Clipping off the fur and covering the animal with mucilage did not prevent dyspnœa, but it took a longer time than usual to heat him up. Here the mucilage acts as an irritant of the sensory nerves and diminishes heat production. It would seem that when heat is applied to the skin that two influences of an antagonistic nature are set up—one to arouse the polypnœic centre, and through it the respiratory, into increased activity; the other, through the respiration and inhibiting fibres of the vagus and probably through the nerves of the skin, a depression of the number of respirations. However, the impulses sent into the polypnœic centre preponderate and mask the antagonistic ones.

These experiments proved that after the removal of the polypnœic centre the fall of respiration frequency by the rise of temperature was due mainly to an irritation of the inhibitory fibres running through the vagus to inhibit the main respiratory centre. These facts still further substantiate the existence of the polypnœic centre. With five centres concerned in the respiratory mechanism, it may be permissible to consider the relation of the thermo-polypnœic centre to the six thermotaxic centres regulating the temperature of the body. As already noted, the polypnœic centre is active in the regulation of the temperature of the body. The thermo-polypnœic centre acts reflexly, but it is ever active. The moment high temperature affects the sensory nerves of the skin this centre signals the medullary respiratory centre to go to work and cool down the body by throwing off heat to counterbalance that thrown on.

As I have already stated elsewhere, the thermotaxic centres located in the cortex are the cruciate and Sylvian and the four basal centres at the base of the brain are also thermotaxic. I have lately made some experiments to determine whether the polypnæic centre and the thermotaxic centre in the gray matter at the most anterior part of the third ventricle are one and the same or different centres. In puncturing the thermotaxic centre at this point, I have frequently obtained polypnœa, with a rise of temperature. In fact the location of these centres is precisely in the same spot, so that I am convinced they are one; in other words, this thermotaxic centre manifests its activity by polypnœa. How the other basal thermotaxic centres stand in relation to the thermolytic centres, which are the respiratory, vaso-motor, and sudorific, is not yet determined. Danilewsky⁶ found that electric irritation of the caudate nuclei had a direct action on the vaso-motor centre, causing sometimes an elevation of arterial tension, at other times a depression; sometimes an acceleration of pulse, at other times a slowing. In another place I have put upon record some experiments to prove that the corpora quadrigemina contain a centre regulating the spinal sweat centres. It is highly probable that part of the basal thermotaxic centres, through an irritation of the sensory nerves, send reflex impulses down the the cord, regulating the spinal thermogenic centres instead of directly inhibiting metabolism.

Some years ago I made experiments with cats, the soles of whose feet were devoid of pigment. They were etherized, the spinal cord bared by trephining, and then divided or at times destroyed *in toto* by a wire thrust between the two trephined openings. Then the animal was heated. It was found, when the spinal cord was divided between the sixth and seventh cervical vertebræ, the right cervical sympathetic divided above the first rib, and the left sciatic cut, that upon heating the right posterior extremity it became more red than the left posterior extremity. Destruction of the cord at this point caused the soles of the feet of

⁶ Pflüger Archiv, Bd. xi., p. 128.

the posterior extremities in the above experiment to be the same in color. These experiments proved that the spinal cord alone has vaso-dilator centres which are called into activity by heat. Now, vaso-dilation and vaso-constriction are means to regulate the temperature of the body. Heat can also excite directly the spinal sudorific centres. Now, sections of the spinal cord or, according to my experiments, of the lateral column alone, are followed, according to the surrounding temperature, by a fall or rise of body temperature.

Ugolino Mosso has shown in curarized animals that strychnia elevates the temperature probably by stimulating the spinal thermogenic centres. Atropin acts in a similar manner, also being accompanied by increased production of heat. Now, it is probable that after section of the lateral columns of the spinal cord the influences of part of the thermotaxic centres have been removed, and the temperature rises and falls according to the external temperature. In this case the vaso-motor fibres have been cut and the blood-vessels are dilated. If, however, a section is made above the main vaso-motor centre, then the regulation of the temperature is much better; the temperature frequently rises. In fact, so able an observer as Heidenhain believed the vaso-motor system to be mainly sufficient for the regulation of the temperature, thus rejecting the idea of thermotaxic centres. Jürgensen found that within the first seven to eight days of extra-uterine infantile life that the body temperature moved within wide limits, and independent of the time of day.

In the case of monsters born without any brain excepting medulla and pons, heat regulation exists, how accurately no one has thermometrically determined. Here the vaso-motor system suffices to carry on the necessary thermotaxis, for no great excess of heat or cold is allowed to test the thermotaxis of infants.

It has also been noted that irritation of the cortex centres was accompanied by a fall of temperature, and their excision by a rise of temperature. Dr. Girard, of Geneva, has recently made some experiments on the same subject,

and finds the Sylvian in the same area, but states that Schreiber found a similar state of affairs after injury to the pons, cerebellum, and peduncles. Now, it happens that I have repeatedly punctured the pons variolii and obtained a rise of a few degrees of temperature for a couple of hours. This is not due to destruction or irritation of a thermotaxic centre here, but is the same phenomenon seen in many parts of the brain, and is due to injury of an afferent nerve of the thermotaxic centres. Now, with the Sylvian and cruciate centres this rise of temperature is not for a few hours, but continues for days, up to the death of the animal. Hence this objection of Dr. Girard's falls to the ground, especially as injuries to any part of the brain are frequently accompanied by a temporary rise of temperature. It seems to me that the Sylvian and cruciate centres are limited in their extent, no other part of the cortex causing a similar state of affairs; that their irritation is followed by a depression of temperature, their excision by a rise of temperature, and compared with the motor centres in the same animals are quite as well circumscribed. Further unilateral excision of these centres is followed by a greater elevation of the temperature on one side of the body than on the other, probably due to a decussation of fibres. All researches go to show that in man more highly specialized thermotaxic centres exist. The cortical thermotaxic areas in man will have to be worked out from pathological observations, for the human cortex attains so high a degree of development that the localization of the thermic points in animals cannot be transferred.

Taking up these four basal thermotaxic centres, Dr. Girard has found a similar disturbance in the temperature curve after their injury by a probe, as I have already demonstrated. He however believes that they are not so circumscribed as I have stated. I have again made calorimetric observations lasting a whole day upon fasting rabbits, both before and after injury of the thermotaxic centres. Usually I selected the hours between seven and twelve A. M. for the normal series, whilst the afternoon hours showed the effect of lesions of one of these basal thermo-

taxic centres upon temperature, H. P. and H. D. The calorimeter error was so reduced that the instrument only varied $\frac{1}{1000}$ of a degree F. per hour for every degree the temperature of the air exceeded that of the calorimeter. A little carpet and sawdust was introduced into the instrument for the animal to sit upon, so as to gradually accustom him to the surrounding copper walls, which absorb heat more rapidly than the animal is accustomed to in the air. The calorimeter used was d'Arsonval's, which has been modified by the addition of an agitator⁷ which could be worked without opening the instrument or hardly disturbing the sawdust.

TO BE CONCLUDED IN MAY NUMBER.

⁷ New York Medical Journal, March, 1889.

A TUMOR COMPRESSING THE MIDDLE LOBE OF THE CEREBELLUM.

By GEORGE J. PRESTON, M.D.,

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in the Woman's Medical College of Baltimore.

TUMORS of the cerebellum, while by no means rare, are sufficiently uncommon to make this case worth reporting. Apology must be offered for the rather unsatisfactory examination of the patient, as he was only seen a few times, death coming very unexpectedly, as often happens in these cases.

Samuel K., æt. thirty-six, white, mechanic. General health had always been fairly good and no syphilitic history. At one time had *colica pictonum*, but no paralysis. About eight months before admission to hospital he began to have pain in back of head and neck, and staggering gait. These symptoms gradually increased in severity. When seen his condition was as follows: Very marked mental hebetude, although could answer questions, in the main, intelligently. Both sight and hearing were almost lost. I regret to say that through some mistake the ophthalmic examination was not made. Pupils moderately contracted and almost insensible to light. Only a slight nystagmus. There was not a trace of any paralysis. Although patient had been ill for so many months, his muscular strength was remarkably good. Sensation was unimpaired. Muscular sense was almost abolished, as shown by various tests, such as putting the limbs in different positions and requesting patient with eyes closed to imitate the position with the corresponding limb, or to search for one hand, which was moved in various directions, with the other. There seemed to be almost total loss of this sense. If one

arm, for example, was raised up to his shoulder and he was requested, with closed eyes, to touch the hand of this arm with his other hand, he would grope about for it as a person would look for an object in the dark. The same thing was true of the legs.

The reflexes were all present, though rather diminished in force. The patient was totally unable to stand erect or walk without assistance. The moment the support was taken away he fell backwards. There was no tendency to fall to either side. Closing the eyes made no difference in the reeling. He complained of pain in the occipital region and the nape of the neck, and this pain seemed to be increased on tapping the back of the head with a percussion hammer. The diagnosis of tumor compressing the middle lobe of the cerebellum was made, and operation recommended, which patient's friends absolutely refused. Death took place very suddenly and unexpectedly. Autopsy made about twenty hours after death showed a cranium unsymmetrical, the asymetry not due to pressure of tumor. Meninges normal. A tumor about the size of a hen's egg, bi-lobed and of rather firm consistency, was observed springing from the posterior portion of the corpus callosum, with attachments to the falx and tentorium cerebelli. The tumor pressed directly on the middle lobe, or worm, and also flattened the corpora quadrigemina. The roof of the mid-brain was soft and somewhat degenerated, as were the optic tracts and nerves. Section of the hardened tumor showed under the microscope a certain number of very fine fibres running through the mass, with a few glio cells. There were present many large round cells, small round cells, and some spindle cells, constituting what might be called a glio-sarcoma. There are two things to be emphasized in connection with this case; the first is the fact of the loss of muscular sense, without any disturbance of motion or sensation, from a tumor compressing the middle lobe of the cerebellum, adding a little testimony to Lussana's theory, announced in 1863, that the cerebellum is the seat of muscular sense. The other point to be observed is that the tumor could in all probability have been removed.

Almost certainly this would have been possible had it been attempted a few months previous, when the diagnosis would have been quite clear. Bennett May, in a case operated on by him in 1887, says he found no very great difficulty in exposing the cerebellum sufficiently for operations in most of its extent. On looking at the case I have reported, post-mortem, I feel sure that the tumor could have been reached and removed.

9 E. TOWNSEND STREET.

PILOCARPINE IN THE CONVULSIVE ATTACKS OF HYSTERO-EPILEPSY, AND IN MANIACAL EXCITEMENT.

By SAMUEL B. LYON, M.D.,

Bloomington Asylum, N. Y. City.

A paper presented to the Council of the American Neurological Society,
September, 1888.

IN the subjects of the hystero-epileptic and kindred neurotic states, which make the victims interesting studies when mentally and morally vivisected, as we see them in the Charcot clinics, it is certainly as desirable to remedy the condition and to restore self-control to the unfortunates, if possible, as to develop typical cases for clinical exhibition. The hystero-epileptic type apparently does not flourish in the conditions of this country as it does in France, but occasional cases do occur; and an epileptic woman who was under my care a few years since was a good subject, and exhibited the capacity to make a very fair exhibition case, had she received the necessary notice and attention. She developed spontaneously the wrapt and striking attitude of prayer, and all the emotional display which accompanies it, in one stage in the *attitudes passionnelles* through which the Salpêtrière subject passes, and would have developed other of the phases had she received any encouragement.

In a case of insanity with some hystero-epileptic symptoms, which was observed by me, there was loss of mental control and abolition of will, with true convulsive movements and marked anæsthesia. Having failed to obtain any permanent relaxation of the spasmodic condition by inhalations of amyl nitrite and ether, an immediate and permanent relief was obtained by the hypodermic injection of the muriate of pilocarpine.

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It is certainly very desirable to have at hand a remedy which can be administered independently of the concurrence of the patient, and which will relax the tension of the nervous force, as pilocarpine is well known to do in the *status epilepticus*, and may be expected to do in the semi-conscious convulsions of hysteria or hystero-epilepsy; and as some volition undoubtedly remains, and the force of habit is by no means abolished, a succession of experiences of the relaxing effects of the drug may turn the tide of impulse and imagination away from its morbid direction and substitute a wholesome impassibility.

There are occasional cases of high excitement among maniacal patients in which a remedy that will temporarily calm them, without leaving the immediate or after ill effects of the powerful sedatives, is much to be desired, and pilocarpine may help us here.

A brief history of the case in which the use of pilocarpine resulted so favorably will be pardoned.

L. H., aged thirty; married about six years; nervous temperament, insane heredity on her mother's side; when first pregnant, at the age of twenty-five, became flighty and talked disconnectedly, but did not reach a high degree of mental excitement; during a second pregnancy she showed in an increased degree the same condition of mental instability as at the first pregnancy; since this time she has at no time been rational, and has been demented to a marked degree; cannot be interested in conversation; wanders about aimlessly; many of her delusions are referable to the sexual system, as that men are plotting to get possession of her, etc.; she has also delusions of persecution, that she has committed the unpardonable sin, etc.; has a foreboding of evil.

The patient has a history of chronic uterine displacement and probable ovarian disease.

Her condition previous to coming here was described as having been both violent and excited, and controllable with difficulty. She was in an absorbed or dazed condition of mind when admitted, and appeared to take little notice of her surroundings. While in the carriage on her way to the hospital she had what her attendant called a fit.

The second morning after her admission she had another convulsive seizure, which had the characteristics of hystero-epileptic attacks. She had marked opisthotonos, insensibility of the cornea, temperature of $103\frac{2}{3}^{\circ}$, pulse 150 and tumultuous, moist skin, mouth open and dry, contractions principally of the neck with the head thrown backward, and violent convulsive movements which seemed excited to some extent by attempts to hold her, and to be not entirely unconscious. She was given inhalations of amyl nitrite and ether, which only temporarily quieted the spasmodic condition; pressure was made over the ovaries with no effect; she was finally given hypodermically one-eighth grain of muriate of pilocarpine over the left ovary, which was soon followed by a profuse perspiration and a fall in temperature to $101\frac{1}{3}^{\circ}$, the convulsive movements ceased, and consciousness gradually returned, which was followed by a quiet sleep. Previous to the convulsive attack her pupils had been widely dilated; during the convulsions they were reduced to one-half their former diameter; during her return to consciousness they were seen to expand and contract quite suddenly, varying with the tension of the general muscular system.

No further convulsions have occurred in the case, but a passive condition has succeeded to her former excitement.

PERISCOPE.

PHYSIOLOGY OF THE NERVOUS SYSTEM.

By DR. GEORGE W. JACOBY.

DE LA NATURE DE LA MALADIE DE PARKINSON, *Journal de Médecine et de Chirurgie pratiques*, Jan. 1889, p. 28.

Dr. Gauthier endeavors to prove that paralysis agitans does not in any manner present the characteristics of the class of affections into which it has been relegated. G. admits that the symptoms are exclusively those of the muscular system, and due to the rigidity of the muscles; also that this rigidity is produced in the muscles themselves, without the intervention of the nervous system. On the other hand, the fact that an analysis of the urine in a large number of cases of paralysis agitans shows an excess in the excretion of phosphoric acid, leads the author to the following conclusions: Firstly, that this phosphaturia is of actual import in the pathogeny of the disease; and, secondly, that as all the symptoms are of a muscular nature, we are dealing with a "muscular phosphaturia," together with a dystrophy of the muscular system.

UN CAS DE TETANOS A DEBUT CEPHALIQUE, AVEC PARALYSIE FACIALE, Drs. Remy and Villar. *Gazette des Hôpitaux*, Dec. 11, 1888, p. 1314.

The cephalic form of tetanus with facial paralysis being of rare occurrence, the authors publish a case of this curious variety in detail. This form of tetanus begins with trismus, usually unilateral, accompanied or followed by facial paralysis of the same side, both the trismus as well as the facial paralysis occupying the same side as the wound. The

patient was injured, on Jan. 7th, by a cut with a knife at the inner part of the orbital arch of the left side, also by a blow with a sand bag upon the left fronto-parietal region. Unconsciousness supervened. Brought to hospital next day. Supra-orbital wound sutured. Two weeks after the accident, when first seen by the authors, he showed ptosis of the injured eye. There was no indication of fracture of the skull. On the injured side, facial paralysis with contraction of the masseter was present. The dental arches were pressed tightly together, and only with difficulty could they be separated a few centimetres. The precise time of onset of these symptoms cannot be determined. During the following days all attempts to open the mouth remained fruitless. Feb. 1st: Stiffness of neck, of sterno-cleido-mastoid noticeable; great difficulty in moving the head; trunk also rigid. The patient cannot sit up nor flex the left leg. Arms not affected. Uninjured side of the face also contracted. Trismus. Respiration became difficult; deglutition affected, contractures increased, and finally tetanic spasms and hallucinations, lasting from Feb. 22d to death, which occurred on March 2d.

The autopsy showed a slight fracture of the orbital arch. No fracture of the base or vertex of the skull. Brain normal. Cord and membranes congested. The facial and trigeminal nerves were examined, but with negative results.

(A complete review of the subject, "Facial Paralysis and Tetanus," by Villar, will be found in the *Gazette des Hôpitaux* for Dec. 22, 1888.)

EPILEPSIE PARTIELLE, DETERMINE PAR UNE TUMEUR CERVEALE, SIEGEANT AU NIVEAU DE LA ZONE MOTRICE. TREPANATION. ABLATION DE LA TUMEURS. DISPARATION DES SYMPTOMES. Péan. *Gazette des Hôpitaux*, p. 189, Feb. 21, 1889.

The patient in this case was a male, aged twenty-eight. At the age of twenty-two, epileptiform attacks appeared, which recurred about every ten days. These attacks became

more and more frequent, until in 1886 they constituted a species of status epilepticus. Bromides kept the attacks somewhat in abeyance. In December, 1888, the convulsions became so severe as to constitute a menace to life. In consequence of a paresis of the right leg, of the age and antecedents of the patient, the diagnosis of a tumor occupying the superior part of the pre and post central convolutions was made. By means of craniometrical measurements the lower end of the fissure of Rolando was mapped out, and from there the location of the upper part determined. At this part a button of bone was removed from the skull, the periosteum being preserved. In the posterior part of the operative field a soft tumor was found, which little by little was entirely removed, with but very slight injury to the cerebral substance. The wound was then closed, and on the tenth day cicatrization was complete. The epileptic attacks became less frequent the day following the operation. At time of publication, two and one-half months after operation, no epileptiform manifestations are present.

IDIOTIE AVEC CACHEXIE PACHYDERMIQUE. DR. GEORGES CONSOT. Bull. de la Soc. Mentale de Belgique, 1888, No. 51, p. 355.

The author agrees with Bourneville and Bricon, that the "cretinoid idiocy" of certain English writers is not cretinism, but actual idiocy with myxœdema. From this point of view, regarding the idiocy as secondary to myxœdema developed during childhood, few cases have been described, but Bourneville and Bricon believe them to be of more frequent occurrence than is generally supposed.

C. adds a new case to those already published. This female patient at time of writing was thirty-one years of age. She was healthy at birth. At age of six months the face began to lose expression and the tongue became voluminous. Growth of body retarded. Swelling and puffiness of skin. Skin, thick, cold, cyanosed, and œdematous; appearance of general anæmia; absence of perspiration; swelling of the mucous membranes of the mouth and tongue. Muscular weakness: intellectual apathy, organic degeneration. The thyroid body could not be felt, although palpation of the larynx and trachea was easy.

Miscellaneous Notes.

DEPARTMENT OF THE INTERIOR. CENSUS OFFICE.

WASHINGTON, D. C., May 1, 1889.

To the Medical Profession:

The various medical associations and the medical profession will be glad to learn that Dr. John S. Billings, Surgeon U. S. Army, has consented to take charge of the Report on the Mortality and Vital Statistics of the United States as returned by the Eleventh Census.

As the United States has no system of registration of vital statistics, such as is relied upon by other civilized nations for the purpose of ascertaining the actual movement of population, our census affords the only opportunity of obtaining near an approximate estimate of the birth and death rates of much the larger part of the country, which is entirely unprovided with any satisfactory system of State and municipal registration.

In view of this, the Census Office, during the month of May this year, will issue to the medical profession throughout the country "Physician's Registers" for the purpose of obtaining more accurate returns of deaths than it is possible for the enumerators to make. It is earnestly hoped that physicians in every part of the country will co-operate with the Census Office in this important work. The record should be kept from June 1, 1889, to May 31, 1900. Nearly 26,000 of these registration books were filled up and returned to the office in 1880, and nearly all of them used for statistical purposes. It is hoped that double this number will be obtained for the Eleventh Census.

Physicians not receiving Registers can obtain them by sending their names and addresses to the Census Office, and, with the Register, an official envelope which requires no stamp will be provided for their return to Washington.

If all medical and surgical practitioners throughout the country will lend their aid, the mortality and vital statistics of the Eleventh Census will be more comprehensive and complete than they have ever been. Every physician should take a personal pride in having this report as full and accurate as it is possible to make it.

It is hereby promised that all information obtained through this source shall be held strictly confidential.

ROBERT P. PORTER, Supt. of Census.

**Which is the Most Powerful ?
and the Most Reliable ?
of All Pepsins ■**

Merck's Pepsin 1:2000 !

— SCALE OR POWDER —

SEE "MERCK'S INDEX," PAGES 106 AND 167

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

THE VELOCITY OF NERVE IMPULSES IN CUT
AND INTACT NERVES.¹

By EDWARD T. REICHERT, M. D.,

Professor of Physiology, University of Pennsylvania.

IT is apparently an almost universally accepted fact that nerve impulses travel along nerve fibres at a rate directly proportional to the length of the fibres, yet it seems, as Schiff has long since pointed out, that this belief is open to serious question; and this is certainly not without some support. The usual method employed in studying this phenomenon is to use the familiarly known nerve-muscle preparation of the frog. There can be no doubt but that in such a preparation, where the continuity of the nerve and spinal cord has been destroyed, the above law holds good, but where the continuity remains intact there are reasons to believe that a different condition of vitality exists. Thus Heidenhain has shown in connection with the "avalanche" theory of Pflüger, that while the same strength of current applied to different points of a cut motor nerve will yield less and less reaction in the muscle as the excitation is farther away from the spinal cord, the nerve when intact will show a gradual diminution at first, then an increase, as the peripheries are approached. The curve of reaction in the cut nerve would then be a *straight* line, in the intact a *curved* line. René has found in experiments on man that

¹ Read before the American Society of Physiologists, December 30, 1888.

the rate of response to sensory stimulus was not proportional to the length of nerve traversed by the impulse, thus it sometimes took less time for reaction to occur when the finger was irritated than when the elbow or shoulder. Hence it is obvious that a less time was required for the impulse to flow over the greater distance. Experiments which I have made in other directions clearly show that not only is the condition of the nerve affected by section, but also that of the centre with which it is connected.

In rabbits, where the motor nerve has been cut, the time of reaction to excitation, other things being equal, is directly proportional to the distance the impulse has to travel; where, however, the nerve is *intact* in many instances reaction will occur in a shorter time from the farther point of excitation—a condition the same as René found with intact sensory nerves in man.

I performed nineteen experiments in rabbits in Schiff's laboratory, Geneva, in which the nerve was left intact. In order to avoid difficulties which would attend the presence of pain or volitional movements, the animals were all anæsthetized, and while in this state transverse section of the spinal cord was made in the lower dorsal or upper lumbar region. The sciatic nerve was exposed at the pelvis immediately above the bifurcation and also near or adjoining the gastrocnemius muscle. The tendon of the muscle was also exposed and cut at its insertion and properly "weighed" and connected with a recording apparatus. This apparatus consisted of two independent electrical circuits recording by suitable instruments upon a revolving drum the time of excitation of the nerve and the time of reaction in the muscle respectively. These may be spoken of as the nerve and muscle circuits. In the latter contraction of the muscle broke a simple metal contact, thus breaking the current, and through the operation of a Dupré chronograph the phenomenon was recorded on the drum. In the other circuit a Du Bois Reymond induction apparatus was placed to regulate the strength of current, also a tuning-fork vibrating and recording upon the drum a definite number of times per second. Between the prongs of the fork was a

metal plug—the two forming a “contact;” thus when the plug was withdrawn the current was “closed” or “opened,” according to the arrangement of the wires, and at the same instant the fork was caused to vibrate. By having the pens on the chronograph and tuning-fork exactly in line, the time interval could be read with great precision to .0001”.

Hard-rubber insulated electrodes were always used, excepting where the muscle was directly excited, in which case pin electrodes were thrust directly into the ends of the muscle. In some experiments one electrode was placed on the sciatic nerve near the pelvis and the other near the muscle, the two being separated by from $5\frac{1}{4}$ to $6\frac{1}{2}$ centimetres of nerve; in others the second electrode was connected with the muscle as just stated.

Opening induction shocks were always employed, generally of medium strength, sometimes of minimum strength and occasionally of maximum strength. The same current was used through both electrodes, a commutator being employed to switch it from one to the other.

In each experiment from four to thirteen observations were made with each electrode, the electrodes remaining throughout the experiment undisturbed. Excitations were alternate, a record being taken by one electrode and this followed in a few minutes by the other, these observations being repeated at intervals of about ten minutes. Thus in each experiment from eight to twenty-six records were made.

In eight experiments both electrodes were placed on the nerve, being separated, as before stated, from $5\frac{1}{4}$ to $6\frac{1}{2}$ centimetres. In three of these *less time* was required for the impulse to travel from the point near the spinal cord to the muscle and cause contraction than from the lower point. To facilitate a clear understanding of the results of these studies the records are given in tabulated form which speak for themselves. In the table which immediately follows, the results of three experiments are given, the number of the experiment is found in the first column, the number of observations at each electrode in the second, the positions of the electrodes in the third, the shortest reaction

time in all of the observations at each electrode respectively in the fourth, the longest interval in the fifth, the average of the sum of all in each series in the sixth, finally in the last column the difference in the average time-interval, which is found by subtracting the figures in the previous column. Thus in experiment No. 1, for example, eight observations were made at each electrode, one electrode being placed on the nerve near the pelvis and the other $6\frac{1}{2}$ cm. lower on the nerve, then are given the time-intervals, lastly the difference in the average time-interval. This last is throughout all the series placed opposite the figures in the preceding column representing the longer intervals, hence we find in this experiment that it required .0099" longer for the impulse to pass from a point near the muscle and cause reaction, than from a point $6\frac{1}{2}$ cm. farther away. In the second experiment it required .0003" longer and in the third .0092" longer for the impulse to pass from the point near the muscle.

FIRST SERIES.

Number of Experiments	Number of Observations	Where Stimulation was applied.	Shortest Time Interval.	Longest Time Interval.	Average Time Interval.	Difference in Average Time Interval.	
1 {	8	At pelvis.	.0169	.0229	.0165		} Maximal strength of current.
	8	$6\frac{1}{2}$ cm. lower.	.0229	.0297	.0264	.0099	
2 {	7	At pelvis.	.0121	.0135	.0820		} Medium strength of current.
	7	6 cm. lower.	.0114	.0148	.0133	.0003	
3 {	8	At pelvis.	.0135	.0148	.0142		} Medium strength of current.
	8	$6\frac{1}{2}$ cm. lower.	.0162	.0256	.0230	.0092	

In the second series the reaction-time was always greater from the farther point. In the five experiments the difference in average time reaction in the first four is remarkably close, while in the fifth experiment (No. 8) the nerve

was doubtless seemingly affected by loss of proper blood supply, and as a result slowness of reaction.

SECOND SERIES.

Number of Experiment.	Number of Observations.	Where Stimulation was applied.	Shortest Time Interval.	Longest Time Interval.	Average Time Interval.	Difference in Average Time Interval.	
4	8	At pelvis.	.0122	.0142	.0131	.0002	} Medium strength of current
	8	5¼ cm. lower.	.0108	.0148	.0129		
5	8	At pelvis.	.0122	.0162	.0140	.0003	} Maximum strength of current.
	8	5½ cm. lower.	.0122	.0169	.0137		
6	6	At pelvis.	.0148	.0155	.0151	.0003	} Medium strength of current.
	6	5½ cm. lower.	.0148	.0148	.0148		
7	7	At pelvis.	.0094	.0118	.0103	.0004	} Medium strength of current.
	7	6 cm. lower.	.0087	.0094	.0089		
*8	7	At pelvis.	.0169	.0291	.0191	.0080	} Medium strength of current.
	7	6 cm. lower.	.0108	.0116	.0112		

*Vessel ruptured while preparing the sciatic, some blood lost; ligature.

In the first series the rate of transmission of nerve impulses cannot, of course, be determined. In the second, however, it is obvious that the rate is represented in the difference in the average time interval, this time being the amount occupied in the passage of the impulse, along so many centimetres of nerve. If we except experiment No. 8, which is obviously abnormal, the average difference in time-interval is .0003". Hence the impulse travels over an average of 5.55 centimetres in .0003", or at the rate of 185 metres per second. This is largely in excess of any published figures, the recognized velocity in motor nerves of mammals being about 34 metres per second.²

² The following is a resumé of the rates of transmission of nerve-impulses obtained by different observers.

Motor Nerves.	{ Lobster—	Fredericq and Van de Velde, 6 metres per second.		
		Marey,	20	metres per second.
	{ Frogs.	Clawson,	21	" "
		Helmholz,	27	" "
		Lamansky,	31	" "
		Bernstein,	32	" "
		René,	20	" "
	{ Mammals.	Marey,	30	" "
		V. Wittich,	30	" "
		Helmholz,	34	" "
		Chauveau,	65	" "
		Jaeger,	26	" "
Sensory Nerves. — Mammals.	{	Marey,	30	" "
		Schellke,	30	" "
		Helmholz,	50	" "
		Richet,	50	" "
		Kohlrausch,	94	" "
		Bloch,	132	" "

In the third and fourth series, one electrode was placed on the sciatic nerve near the spinal cord, and the other in the muscle. Out of eleven experiments thus made, in seven less time was required for reaction from the electrode near the cord than when the muscle was directly excited. These seven constitute the third series.

THIRD SERIES.

Number of Experiment.	Number of Observations.	Where Stimulation was applied.	Shortest Time Interval.	Longest Time Interval.	Average Time Interval.	Difference in Average Time Interval.	
9 {	6	At pelvis	.0103	.0135	.0123		Medium strength of current.
	6	muscle.	.0216	.0297	.0223	.0100	
10 {	11	"	.0125	.0148	.0120		"
	11	"	.0125	.0162	.0131	.0011	
11 {	11	"	.0108	.0138	.0101		"
	5	"	.0131	.0162	.0139	.0028	
12 {	13	"	.0095	.0118	.0107		"
	13	"	.0125	.0149	.0132	.0005	
13 {	10	"	.0088	.0101	.0092		"
	10	"	.0088	.0108	.0094	.0002	
14 {	8	"	.0081	.0102	.0082		"
	8	"	.0088	.0108	.0094	.0012	
*15 {	7	"	.0095	.0108	.0101		"
	7	"	.0095	.0108	.0104	.0003	

*Animal in an exhausted condition from hæmorrhage following section of the cord.

In the fourth series, in which are included the remaining four experiments, a longer time was required for the passage of the impulse for the electrode on the nerve.

FOURTH SERIES.

Number of Experiment.	Number of Observations.	Where Stimulation was applied.	Shortest Time Interval.	Longest Time Interval.	Average Time Interval.	Difference in Average Time Interval.	
16 {	9	At pelvis muscle.	.0121	.0162	.0149	.0006	} Maximum strength of current.
	8		.0121	.0175	.0143		
17 {	5	"	.0101	.0129	.0110	.0006	} Medium strength of current.
	5		.0094	.0094	.0094		
18 {	10	"	.0089	.0115	.0088	.0008	} "
	10		.0075	.0089	.0080		
19 {	7	"	.0094	.0118	.0103	.0008	} "
	4		.0088	.0088	.0088		

Here, as in the second series, it required a longer time to pass from the upper electrodes; and also, it will be noticed, the differences in average reaction times in the four experiments are remarkably uniform. Approximately about $7\frac{1}{2}$ centimetres of nerve intervene between the upper electrode and muscle; the difference in average reaction is, on an average .0007". Assuming that the time of reaction is the same when the stimulus is applied directly to the muscle, or to the nerve at its entrance to the muscle, the impulse would travel along $7\frac{1}{2}$ centimetres in .0007", which would be at a rate of about 107 metres per second. This is much slower than what was found in the second series. Differences in the nature of the two series of experiments will doubtless explain this—in one case the nerve being excited near the muscle, and in the other the muscle itself, it requiring less time for reaction in the latter instance.

It is obvious from the above experiments that the whole subject is open to extensive and careful research.

PHYSIOLOGICAL LABORATORY,
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SOME POST-HEMIPLEGIC DISTURBANCES OF MOTION IN CHILDREN.¹

BY PHILLIP COOMBS KNAPP, A.M., M.D.,

Clinical Instructor in Diseases of the Nervous System, Howard Medical School; Physician to Out-Patients with Diseases of the Nervous System, Boston City Hospital.

IN a paper² which I had the honor of presenting to this Association a year ago, I spoke of the frequent occurrence of post-hemiplegic disturbances of motion in cerebral infantile paralysis. Certain forms of these disturbances have been carefully studied and are well known, among them being athetosis and post-hemiplegic chorea; other forms, such as ataxia, post-hemiplegic paralysis agitans, and post-hemiplegic pseudo-sclerosis, are less common. It is not my purpose, however, to speak in detail in this paper of these various forms of post-hemiplegic disturbance of motion. The exhaustive treatise of Greidenberg³ renders such a task unnecessary, but it may be well to cite for reference the elaborate classification which he makes of these phenomena—the most thorough schedule that has yet appeared.

Contractures	Apoplectic	Spasms	Clonic. Tonic. Intermitting. Muscular rigidity.
	Early, Paralytic, passive, temporary.		
	Late	Constant, continuous, fixed, changeable (latent).	
Increased tendon reflexes.			
Associated movements.			
Tremors	Essential	Reflex clonus.	Tremor proper. In the form of paralysis agitans. In the form of disseminated sclerosis.
Hemichorea		Constant.	Mixed forms in different combina- tions.
		On intended movement—disturbance of co- ordination (hemiataxia).	
Athetosis.			

¹ Read before the American Neurological Association at the First Triennial Congress of American Physicians and Surgeons, 20th September, 1888.

² Hemiplegia in Childhood. JOURNAL OF NERVOUS AND MENTAL DISEASE, August, 1887.

³ B. Greidenberg. Ueber die posthemiplegischen Bewegungsstörungen. Archiv. f. Psychiatrie, xvii. 131, 1886.

The following cases present a form of post-hemiplegic disturbance of motion, which, on analysis, can be made out to be a combination of some of the forms in Greidenberg's table, but which have not yet been thoroughly described.

Observation I.—George E., three and a half years. First seen June 15th, 1887. History of epilepsy in mother's family, and his mother herself is epileptic, having had two convulsions during pregnancy. He is the first child; was born after an easy labor of twelve hours' duration, without the use of instruments, the head presenting. No history of injury. He was a backward child, and did not sit up until the age of two. At present he can stand and walk a step or two with support, but usually does not attempt it, getting about by creeping. He has had one attack in the night, probably convulsive, of which no details could be obtained. Ever since he began to use his limbs his mother has noticed that he could not use his left arm well, and that his right leg was not as good as the left. He is said to be bright, to be able to sing with fair correctness, to repeat little verses, and to have a good memory. The speech, however, to a stranger is quite indistinct. He breathes through the mouth, drools constantly, and has some slight difficulty in swallowing. He is subject to colds, as the clothing over his chest is constantly wet from his drooling. The appetite is dainty, and he has a delicate stomach. He still soils himself occasionally. The child is well developed, with no deformities, the two sides being symmetrical. The head is nineteen and three-quarter inches in circumference. The left leg and arm are held rather rigidly, and there is inco-ordination when he tries to use the left hand, the movement being awkward and jerky, but there are no involuntary movements when the arm is at rest, with one exception. When he makes a movement elsewhere, there is often an associated movement of the left arm. The arm is rotated inwards, extended at the elbow, flexed and pronated at the wrist, and flexed at all the phalangeal joints. Individual movements of the arms, hands and legs seem fairly well done, except (dorsal) extension of the right foot. Passive (dorsal) extension of the left foot reveals a slight

constant rigidity. On attempting to walk, he walks with both feet inverted, and the right leg at times gives out. The left forefinger is usually kept clenched in the palm. Sensation, reflexes and electrical reactions showed nothing unusual. There was nothing abnormal discovered in the movements of the face and eyes. He was referred to Dr. F. H. Hooper, who found a narrow, high-arched palate, and a mass of adenoid vegetations in the naso-pharynx, which were removed, relieving the drooling considerably. In September he had a convulsion, which his mother attributed to indigestion. In October his general health was better, and he got about better. In October there was considerable inco-ordination of the left hand and a little of the right. The left hand was fairly strong. The gait was unsteady, the right foot being twisted after him. The associated movement above described persisted, and he still drooled somewhat. He crept rapidly, jerking his legs under him, and throwing his feet up in the air. January 4th, 1888, the condition was practically unchanged. He was beginning to walk a little, and his movements were less irregular and stronger. The inco-ordination and associated movements persisted. Some months later I received a letter from the family physician, telling me that the child had had more convulsions, still attributed to indigestion.

Observation II.—Grace S., seven years old. First seen May 11, 1887. Family history negative; first child. The labor was protracted and difficult, lasting from Tuesday until Friday. Forceps were used; the head after delivery was out of shape, and for some time there was a great question about resuscitating her. Her physician told me that she was nearer dead than any child he ever saw that recovered. She was always a large fat child. At six months she had diarrhœa, and after that she could not sit up like other children. She sat alone at two, crept at three and a half, talked at four, and began to walk alone at five. About two years ago she began to have attacks at night, in which she became rigid, moaned, and frothed at the mouth, the arms twitched and the right arm shook. After this she could not keep still. She never had more than twelve of

these attacks. Her left side has always been the most affected, and she can use the left arm but little. When she does not feel well, the left hand will sometimes open and shut involuntarily. She is reported to be unusually bright in many ways, to have an excellent memory and a large "bump of order." She notices quickly, and likes to help about the house. Lately, for no reason that she can give, she cries when left alone. She now sleeps well, but until she was three or four years old she slept very little, waking and screaming, apparently from pain. She talks a great deal, although her speech is nasal and very indistinct, but she understands what is said, and never uses the wrong word. She drools constantly, and takes cold easily. She breathes through the mouth, which is constantly kept open. She can swallow only liquids and soft food. When about five years old she had massage, after which she began to walk. The child is well-nourished and well-developed, the left arm being a little larger than the right. The left parietal region is more prominent. There is right external strabismus. The open mouth and the constant drooling give the child the appearance of an idiot. The right disk is smaller, and there is a lack of pigment, but no vascular changes. She can move the left arm but little, and the hand hardly at all, and the movements of the right hand are uncertain and not co-ordinated. The left arm is rigid, and kept rotated inwards and carried a little backwards, hyperextended at the elbow, flexed and pronated at the wrist, and the fingers flexed over the thumb, which is between the fore and middle fingers. This rigidity relaxes somewhat at rest, but is heightened by any attempt at motion of either arm, or by excitement. Motion of the left hip and knee is fairly good; at the ankle there is slight motion, but the foot is held with some rigidity in equino-varus. The gait is peculiar; she walks on the left toe, the foot being inverted, and the left leg is weaker, giving out occasionally; the right foot goes first, and the left leg is dragged after it, crossing the right, while the body is thrown forward at the hips, and progress is toward the right. On exertion or excitement there is also some rigidity of the right leg. She

uses the right hand very slowly and awkwardly to pick up things, and the effort brings on associated movements in the left side—either the spasm of the arm above described, or a similar spasm, except that the hyper-extension of the elbow is replaced by semi-flexion, or the hand is opened. The toes of the left foot are also extended (dorsally) and the inner edge of the foot is turned upwards—a form of associated movement to which Strümpell⁴ has called attention. There is also great inco-ordination of the left hand, which she cannot open herself; but at times it opens on associated movements. When opened passively, the hand shuts slowly. There is less loss of power than inco-ordination. The tongue is protruded with difficulty. The knee jerks are little, if at all, exaggerated; there is no ankle clonus; the other reflexes and the electrical reactions show nothing unusual. As in the former case, Dr. Hooper found adenoid growths, which he removed, causing a marked improvement in the drooling, speech, swallowing and general expression; the child ceased to look like an idiot. Massage and light gymnastics have aided the other conditions somewhat.

Observation III.—M. L., female, six. Seen with Dr. Boland, of South Boston, October 7, 1886, and January 22, 1887. No nervous heredity could be discovered, and the family history, so far as known, is good. The child has been in the charge of a very intelligent woman, who has observed her with great care. During pregnancy an unsuccessful effort was made by the mother to induce an abortion, which she now thinks the cause of the child's condition. Labor came on at term, without any accident. For the first nine months the child is said to have cried most of the time. At no time has there been a history of any illness more than the ordinary complaints of childhood. Since nine months of age, she has been in her present condition of constant muscular rigidity. She is unable to stand without support, but when held up by the arms she can bear

⁴ A. Strumpell. Ueber einige bei Nervenkranken häufig vorkommende abnorme Mitbewegungen im Fusse und in dem Zehen. *Neurolog. Centralblatt*, 1 Jan., 1887.

most of her weight on the feet, although she says her left leg is "n. g." (no good). Examination showed a dislocation of the left hip, probably congenital. She presents no deformity or muscular atrophy; the muscles are well developed, and even slightly hypertrophied. Her nurse states that at times the muscular rigidity ceases for a minute, and she becomes more limber. The rigidity is greater in the morning and in bad weather. She can neither stand nor sit, but either lies on the floor or is slung by the arms to a spiral spring in the doorway, which she enjoys very much. Any movement, touch, or excitement exaggerates the spasm. At times she has crying and screaming spells. She can move her head very well, and can roll over. She will not stay on her back, and can be put there only with difficulty. If put on her back, she rolls over to the left side with some difficulty, and gets on her belly, which is almost the only voluntary movement, except of the head, which she makes. She does this when told to, as well as at other times, but the reverse process, from the belly to the back, cannot be performed. She sleep on her belly, turning her head to the left, so as to lie on the right side of her face. During sleep she is very apt to raise the left arm from the bed, letting it fall with such force upon her face as to hurt herself, so that her nurse ties that arm to the side of her crib. At times she gets a little blue about the lips. Except for occasional attacks of diarrhoea she seems well, and makes no complaint. She eats and sleeps well; she has perfect control over her sphincters. Mentally she is said to be unusually bright. Her speech, however, is very indistinct, so that it can be understood only with difficulty. The trouble, however, is purely one of motor inco-ordination, for she talks readily, and always uses the proper words. Her disposition is remarkably sweet, and she is, except when she has a crying spell, of a very sunny and even temper. She has not been taught to read, but her memory is very good. She learns the words of little poems in her picture books on two or three repetitions; she enjoys music, catches the words and airs of the popular songs of the day, and can sing them herself in good tune

and time. She knows and can name and distinguish the ordinary colors, takes a lively interest in what is going on about her, remembers me and calls me by name after three months. She enjoys watching the people on the street and the distant landscape, and seems to appreciate the beauty of a sunset. She is docile and obedient. The child is well developed, well nourished, the muscles a trifle hypertrophied. There is dislocation of the left hip. The vision is good, the pupils react to light, and the field of vision and ocular movements are normal. She keeps the mouth open, but can close it, and she does not drool. The tongue can be moved naturally in every direction. She has a habit of raising and lowering the under jaw rather stiffly, and of nodding the head slowly, especially when talking or excited. She can twist her head and shake it from side to side, but, as a rule, keeps it pretty well back. The body is kept rigid, the arms extended in the crucifixion attitude, her legs rigidly extended, the feet extended, inverted, and in (plantar) flexion,—the spastic position. On lifting her up and requesting her to touch an object, she seems to have a little power to move the arm backward from the plane of the body, but this, and the rolling over on to her belly, are the only voluntary movements, except of the head, which she can make. She never keeps her arms by her side. If anything is put into her hand the fingers close round it tightly, but without any voluntary action on her part—much as a little baby will grasp your finger. The entire body is kept in a ramrod-like rigidity, so that if you want to make her sit up, you must double her up by force, like a spring-bladed jack-knife, and hold her in position, so that she will not straighten out again. This same resistance to passive motion is noticed in all the muscles. At times she is limber enough to be made to bend the hips comfortably to sit in her nurse's lap, but any excitement or touch brings on this ramrod-like rigidity. Any attempt at passive motion is resisted with great strength. At times, however, the limbs seem to move, independently of the will, in a very awkward fashion, although with considerable power. The foot, for instance, is everted, the arms move,

the legs are drawn up, but none of these movements can be performed by the will, and they all show marked inco-ordination. The sensation is normal. The muscular spasm was such as to render any attempt to test the reflexes nugatory. The same may be said also of the electrical tests, although it was possible to get a response from the deltoid to the faradic current. The application of the poles elsewhere produced such an exaggeration of the spasm as to prevent the reaction.

In these three cases we have a cerebral infantile paralysis—a double hemiplegia in the last,—with a rather definite group of motor disturbances. The cases differ markedly, of course, from the forms of hemiplegia with late contracture and atrophy which I reported in my former paper.

This form of motor disturbance is unlike athetosis or post-hemiplegic chorea in that the spontaneous movements are rare; the phenomena are excited wholly or chiefly by intended movements, and the characteristic athetoid or choreic movements are absent. It differs moreover from the post-hemiplegic ataxia of Grasset⁵ in that other elements beside ataxia go to make up the picture. In Observation I., for instance, we see first a tonic spasm of the paralyzed side, increased by excitement, by efforts to use the affected side or even the other side, thus coming under the heading of a true associated movement. In Observation II. the character of an associated movement is so pronounced that we even find special forms of it described by Strümpell. This tonic spasm is certainly akin to the contracture of ordinary hemiplegia, yet it is, if I may be allowed the expression, more “active” than contracture, and less fixed in its type. In late contracture, there is a passive, fixed shortening of the muscles, which does not vary; this, on the contrary, resembles more the active contraction of voluntary movement. In addition to the tonic spasm and the associated movements, there is also, in Observation I. and II., a distinct loss of the power of co-

⁵ J. Grasset. D'une variété non décrite de phénomène posthémiplegique (Forme hémia-taxique). *Le Progrès Médical*, 13th Nov., 1880.

ordination. In Observation III. the spasm predominates; associated movements are thrown so far in the background as almost to escape notice, and the spasm has so far overcome any volitional power over the muscles that inco-ordination is also a subordinate factor.

This symptom-complex—tonic spasm, associated movement, and inco-ordination—although, of course, merely one of Greidenberg's "mixed forms," seems to be a fairly distinct type of post-hemiplegic disturbance of motion. It is not very uncommon, and in my search through the literature of the subject I have found various cases reported under the headings of chorea or athetosis, notably by Fletcher Beach,⁶ and also by Clay Shaw⁷—"imbecility with ataxia." Gowers, moreover, has described a form of slow, mobile spasm following hemiplegia which is closely akin to this. As he first described it,⁸ the spasm was involuntary, but it was exaggerated on motion. Since then he has given a fuller description.⁹ "The most common form is that in which there is tonic spasm, slowly varying in relative degree in different muscles, and thus causing slow, irregular movements, chiefly conspicuous in the hand, and slow, irregular inco-ordination. From this character it may conveniently be termed 'mobile spasm.' It is commonly conjoined with more or less permanent rigidity, which tends to fix the limb in a certain posture. This spasm may cease during rest, but it is renewed by any attempt at voluntary movement, which is disordered by the spasm, being rendered ataxic or inco-ordinate. With this slow, mobile spasm are often associated other involuntary movements, which may persist when the limbs are at rest, and may assume the character of athetosis or chorea. This type differs somewhat from the type I have described in being apparently more variable, and in having less associated movement combined with it.

⁶ Fletcher Beach. On Cases of Athetosis. *British Med. Journal*, 12th June, 1886.

⁷ T. Clay Shaw. On Athetosis, or Imbecility with Ataxia. *St. Bartholomew's Hospital Reports*, ix. 130, 1873.

⁸ W. R. Gowers. On "Athetosis" and Post-hemiplegic Disorders of Movement. *Medico-Chirurgical Transactions*, lix. 219, 1876.

⁹ W. R. Gowers. *A Manual of Diseases of the Nervous System*, ii. 79, 1888.

Of the pathology little can be said. Gowers thinks that his "slow, mobile spasm" is especially common in those cases of meningeal hæmorrhage which he calls¹⁰ "cerebral birth palsy," but certainly it cannot be regarded as pathognomonic of a hæmorrhage, as the evidence of difficult parturition is lacking in some cases. I am not disposed to regard this condition as having any localizing character, not even as pointing to a lesion near the thalamus. In Observation I. and II. I am disposed to think the lesion was cortical, but I have seen this tonic spasm with inco-ordination as a transitory system at times in cases of ordinary chorea where the symptoms were unilateral. I think that the most that can be said at present is that this "mixed form" of tonic spasm, inco-ordination, and associated movement, like most if not all of the other forms of post-hemiplegic disturbances of motion, points simply to a lesion of the pyramidal tract which either deranges the initiation of movement in the motor centres, or impairs the conduction of the motor impulse in the nerve fibres.

¹⁰ W. R. Gowers. Clinical Lectures on Birth Palsies. *Lancet*, 14, 21 April, 1888.

PRIMARY DEMENTIA, WITH DESCRIPTION OF TWO CASES.

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THE comparative infrequency with which this disease of the mind is met with is sufficient to account for the meagreness of the literature that is to be found bearing upon this subject. The study of the works of authors who wrote on insanity in the earlier days of medicine, will show the convincing fact that the essential symptoms of this form of mental alienation were recognized and studied at that time, and it is further noticeable that a vast nonuniformity of opinion existed as to what the proper designating title of the disease should be.

This disease of the mind was first described by Esquirol, and called by him acute dementia. Pinel regarded it as a kind of idiocy, and named it stupidité. By some others it was regarded as constituting a form of melancholia. Of the more recent writers on insanity, Dr. Hammond, in his excellent "Treatise on Insanity in its Medical Relations," refers to it at some length, and calls it primary dementia, which seems to be the most fitting designation, since we are accustomed to associate the word acute with a disease sudden in its origin, while the development of primary dementia may extend over many weeks, and even months. A careful study of the statistical tables contained in the annual reports of half a dozen representative asylums, located in the Northern, Middle, and Southern States of this country, show that, out of more than 1,300 admissions into these institutions during the year 1888, 29 were diagnosed as being cases of primary dementia. Sex seems to be an inconspicuous factor in the etiology of this disease, since the number of occurring cases was about equally

divided between male and female. The ages of those affected are not given, but it seems to be most frequently found in youth and early adult life. Although we are, as yet, on the borderland of the study of this affection, and, I may say, completely ignorant of the pathological changes, if there be any, that mark the inception of it, or arise later on in the course of this disease, we do know that an apparently complete restoration to mental health and activity frequently marks its termination. In view of this fact, some of the German authorities adopted the seemingly more appropriate term of primary curable dementia, but even this was by no means universally accepted; for it was argued that the employment of the word dementia as we use it, within itself, precluded the possibility of an ultimate recovery; and such, as observation and experience have shown, was not the case. The satisfactory study of primary dementia is difficult, from the fact that the opportunities for clinical observation so rarely present themselves, and even then, when such cases are admitted into an asylum, the disease has so far progressed that the study of its most interesting stages has been lost; and it is frequently impossible to obtain any definite knowledge of the mode of its inception and subsequent course of development from those who may have had the patient in charge. But this fact is insignificant in the face of the greater problem we must solve before the study of the human mind in disease can be reduced to anything like an exact science, by reason of a knowledge of its composite qualities; before we are familiar with the causes of the commonest phases of mental action, and why they are so. Many ingenious, and, it may be said, some fanciful theories have been promulgated and expressed regarding the inherent composition and qualities of the mind. These need not be discussed or reviewed here, and mention will only be made of the physical media theory, in order that the basis for the admission of primary dementia as a specific affection may be more clearly presented. Those who defend the physical media theory say that the mind is a distinct derivative of certain organic parts, and depends entirely for manifestations of its normal power

upon the constant integrity of those parts. An intimate relationship is recognized between mind and body; a relationship that has, on one hand, certain structural elements of the human organism, and, on the other, certain manifestations of mental actions. If, then, this be the relation that exists between mind and body; that the evidences of mental action as the outcome of a generic force that proceeds apparently from the so-called physical media, constituted here of cells of nervous matter somewhere in the brain, it seems both as fair and as reasonable to assume that such a disease as primary dementia may as easily exist, providing conditions favorable to its creation are present, as melancholia or mania. That operating influences strictly of a mental character, so far as we can see and believe, do exert their power in the production of such diseases as melancholia and mania we have ample evidences so modified, and, I may add, perhaps intensified, that the nature of the resulting disease may be changed? The variations in vitality and normal integrity of the cells that constitute the so-called physical media, and their power of resisting impressions that abnormal influences may make upon them, are as diversified as the influences themselves. It is always an important object in the study of diseases of the mind to determine, if possible, the nature of the cause, or causes, responsible for such conditions, also the duration of the action of such agencies, and the susceptibilities of the individual to such influence. This seems specially desirable in cases of primary dementia, for an intimate knowledge of the earlier stages of the disease in such cases will aid us largely in making a tolerably sure prognosis as to what the future outcome of the disease will be.

There seem to be two distinct forms of this affection, and the essential difference between them lies in the time consumed in the evolution of its consecutive stages, plus one or two mental symptoms, as displayed chiefly in the physiognomy of the person affected. Blandford and also Tuke make mention of the two forms, only in so far as the time consumed in the complete development of the disease, and the comparative chances of recovery.

In one form the disease is abrupt in its invasions, and so quickly does the change come that there seems to be a complete paralysis of every mental manifestation. In the second variety, the disease is so gradual in its invasion that the decay of mental powers cannot be appreciated from day to day, and it is only after many weeks that the result of its work can be clearly seen. As illustrating more fully the two forms of this disease, I will relate the history of a case of either form, both of which were admitted in this asylum during last year.

The first case, C. H. S., a young, well developed male, eighteen years of age, a clerk by occupation, and possessing a liberal academic education, was admitted here in May, 1888. It was stated at the time of his admission that many of his ancestors were insane, including a grand-uncle and aunt on the mother's side, and a grand-uncle on the father's side. His father exhibits many pronounced eccentricities, but may not be called insane. This young man's physical health had been good all his life, and his mind and body had both been well exercised from his boyhood. In disposition he was always cheerful, pleasant and kind, and a model member of a religious organization. Upon completing his academic course, in his eighteenth year, he began the active business of life, and, by persistent zeal and industry, made commendable progress in it. He seemed perfectly well in every respect until one evening, when attending a meeting of the religious body he was a member of, he became involved in a quarrel with a fellow-member, his best friend, and blows were passed between them. He immediately left the meeting, returned to his home, and told his father of his deep mortification and disgrace at having struck his best friend. He then proceeded to make a number of charges against himself, such as immorality, and so forth. From that hour he was changed. He became quiet and morose, and on the second day after his trouble began he had a mild maniacal outbreak, and fancied the house was on fire. This delusion soon left him, and he fell into a state of complete apathy. He would sleep for hours at a time, and developed a ravenous appetite. He

lost all interest in home affairs, and seemed to forget even the names and faces of his immediate family. When admitted to the asylum a few days later, he presented the mental characteristics of a dement. On addressing him, he would sometimes fail to notice you at all, and, at other times, he would show sufficient interest to stare blankly at you for many seconds ; and then perhaps reply, but only in monosyllables. Upon shaking his hand, you grasped a cold clammy object, that gave no response to the pressure from your own. The extremities were all cold ; his face had a heavy look ; and the pupils were widely dilated. The entire bodily circulation was poor. He had no delusions, and was negligent and slovenly in the care of his person. All the while he retained a vigorous appetite, being led to and from his meals by an attendant. In every respect he led rather a negative existence, with barely sufficient visible mental operations going on to warrant the belief that his mind was not wholly dead to external stimuli. He remained in this condition for about seven weeks, being treated in the meanwhile by administration of various tonic drugs, and endeavoring to interest him in some light form of labor. At the end of this time, almost one by one his faculties seemed to return ; he began asking about his home and friends ; became more tidy in the care of his person ; and, just from that time on, he rapidly grew better.

Just here it might be interesting to mention the remarkable revolution that took place in his disposition during his mental enfeeblement. It was stated in the beginning of this history that he was cheerful, pleasant and kind in disposition, and of a religious turn of mind. After his recovery, all the finer feelings of his nature seemed blunted or destroyed. He no longer took any interest in religious matters, and indulged freely in the use of profane language. His entire disposition seemed to have been moulded and shaped anew during his illness. It is possible that the attack only brought to light what lay dormant in his nature. In May, 1889, he was discharged recovered, and remains to-day in vigorous mental health, with the only peculiarity of this perverted condition of his former disposition.

The second case, and the one constituting the other form of primary dementia, is that of E. G., a young woman, twenty years of age, fairly well educated, and a seamstress by occupation. Little of interest could be learned of her family history, save that both parents were living, and that her father had been insane a number of years, "from the cause of sickness." It is stated that the daughter resembles the mother both mentally and physically. This young woman had no marked personal peculiarities, beyond that she was of a nervous temperament, and was said to be refined in her tastes and cheerful in her disposition. When eleven years of age she had an attack of typhoid fever, which left her hearing slightly impaired, but from that time until the beginning of her mental trouble, two years ago, her health was good. At the beginning of her mental disorder, she passed through a period of mild depression lasting about five weeks. Being fond of pleasures, and of a social disposition, it was thought that her close confinement at work, and consequent inability to indulge her tastes in this direction, brought on this melancholic disposition. Being induced to make a change in the scenes of her daily life, she did so, and visited friends in another State, only to return shortly after to her home worse than when she left it. Shortly after this, the fixed look of depression that had settled upon her face began to fade from it, and in its stead came a less fixed and variable one. She began to do and to say silly things, and seemed to be losing all interest in her home and friends. At one time she had a mild attack of excitement, but it was of short duration. All habits of neatness, and her former sense of modesty and refinement deserted her. The period covering the full development of the disease embraced about eight months, and there was a gradual loss of mental power from the first, without any conspicuous and fixed symptoms of any other mental disease. Little more than a year after her attack began she was admitted to the asylum, and is here yet, a total dement. These two cases seem to me to fairly illustrate the two forms of primary dementia; and I give them, not as constituting strict exponents of any accepted nomenclature or

classification, but, as alienists might regard them, for practical purposes. The diagnosis of the first form of primary dementia may be difficult at times from its close resemblance to some types of melancholia, but as the symptoms of either are accurately defined and compared in standard works on insanity, I will not attempt to state their differentiating features here. A little patience and close observation will enable us to arrive at proper conclusions as regards the latter form.

MORRIS PLAINS, N. J., July 1, 1889.

THE THERMO-POLYPNŒIC CENTRE AND THERMOTAXIS.⁸

BY ISAAC OTT, M. D.

[CONCLUDED].

THESE hourly calorimetric observations brought out plainly the fact that normally there was no necessary relation between temperature and H. P., that the H. P. is constantly fluctuating up and down with the H. D. This was shown much more readily in man, whose temperature did not alter much in the calorimeter, yet whose H. P. fluctuated considerably. Insults to the basal centres with either small or large probes or hollow tubes, proved that these injuries neither incite nor inhibit H. P. as a necessary consequence, but simply disturb the relation between H. P. and H. D., frequently causing increased temperature, which is also at times accompanied by H. P. temporarily increased more than H. D. This temporary increase of H. P. had no necessary relation to the temperature increase, for the temperature may rise and H. P. be diminished (Fig. 5), or the temperature may fall and the H. P. be increased (Fig. 6). In my hourly observations on septic fever, the rise of temperature in the beginning was usually accompanied by a H. P. increased more than H. D. for a few hours; yet the fever has no necessary relation to the H. P., for in exceptional cases H. P. was diminished and the fever continued as usual. These facts go to prove that these six thermotaxic centres have no necessary relation to the H. P., either to increase or diminish it; all they do is to preside over the relation of H. P. to H. D. and preserve the normal temper-

⁸ Read before the New York Neurological Society, January meeting.

ature at a fixed point. If these centres are injured by probes or tubes, large or small, or by septic poisons, albuminoids, peptones, papayotin, or neurin, then they lose their regulating power, and the relation between H. P. and H. D. is changed, and the temperature usually increases, although exceptionally may decrease. As I have proved that it is upon the basal thermotaxic centres that the fever poison mainly acts, it follows that the great interest to determine whether H. P. is increased or diminished in fever is not of

Exp. 224.

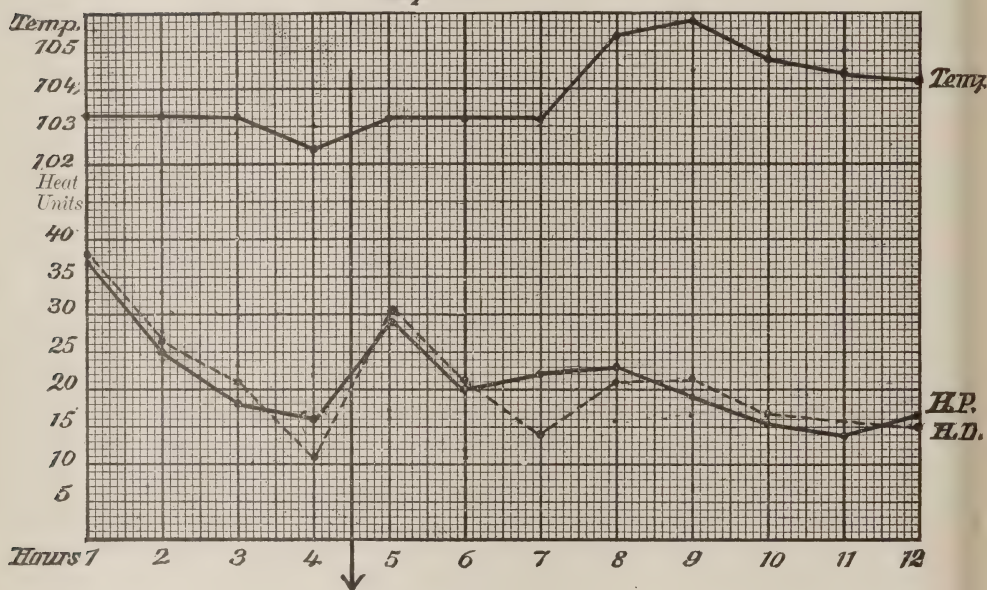


Fig. 5.

Puncture into thermotaxic centre about the gray matter at most anterior part of third ventricle.

so much account; for usually in the setting up of fever, heat production is increased temporarily more than heat dissipation; there is no necessary relation between heat production and the fever, or even the temperature. The vital part affected in fever, as regards the temperature section of it, is a disorder of the thermotaxic centres, which means a disorder of the relation between heat production

and heat dissipation. Liebermeister, in 1875, in his "Pathologie des Fiebers," had a clearer conception and was nearer to the true explanation of fever than all subsequent experimentalists. In the same way, when antipyretics are given to an animal, and the heat production, heat dissipation, and temperature are studied for some hours before and after the drug, it is found that the depression of temperature is often accompanied by a temporary decrease of heat production

Exp. 236.

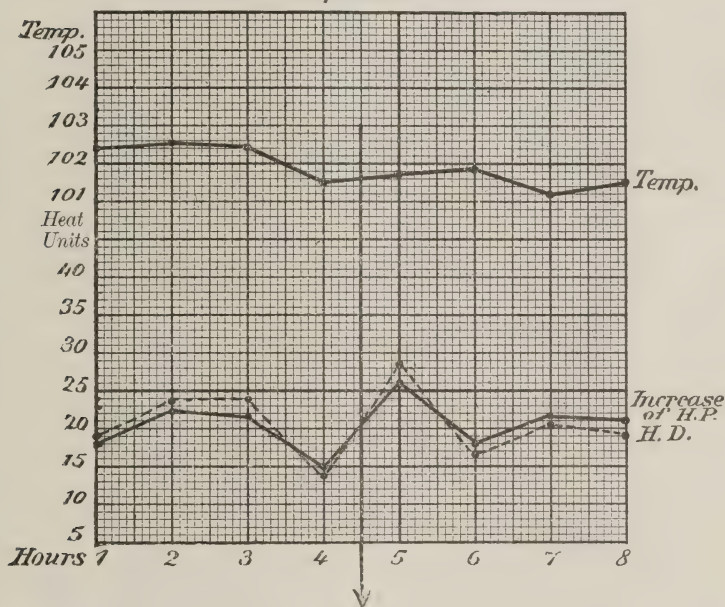


Fig. 6.

(Fig. 7); but this decrement of heat production has no necessary relation to the fall of temperature; for I have often seen the temperature fall and the heat production increase (Fig. 8), or even the temperature increase and the H. P. fall (Fig. 9).

Prof. Chittenden, of Yale, has studied the effects of quinine and antipyrin upon rabbits who have fasted three days, and noted during the whole day the amount of carbonic acid exhaled both before and after the antipyrin. He

arrived at the conclusion that quinine exercises at the most only a very slight depressing influence upon body temperature, and has but a minimum effect upon the production of carbonic acid. Antipyrin in therapeutic doses has no special influence upon the production or elimination of carbonic acid. Lately he has found antipyrin to diminish urea and uric acid. Kummagawa has recently studied the effect of antipyretics upon the nitrogenous tissue changes. He brought dogs to a nitrogen equilibrium by means of a definite amount of food, and then in addition to their daily diet

Exp. 225.

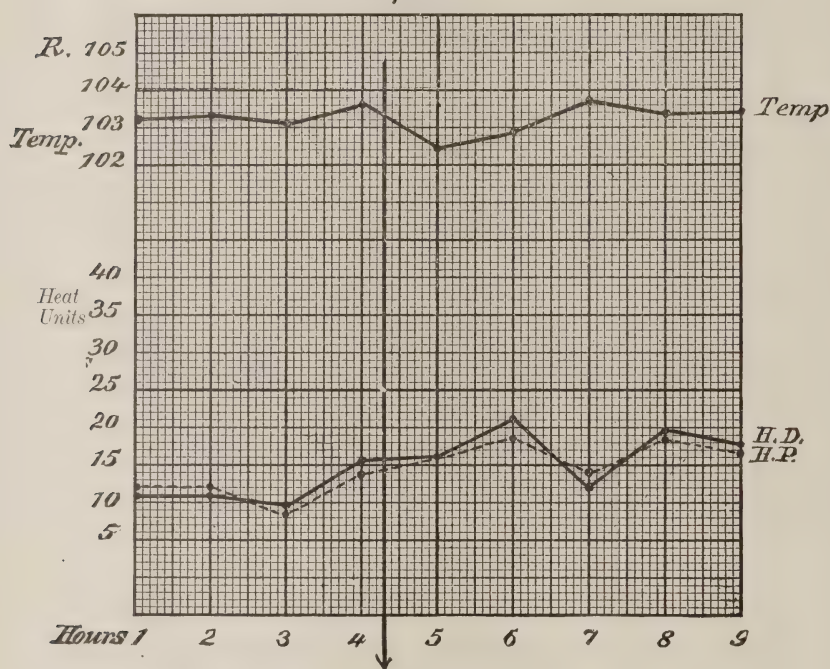


Fig. 7.

gave them antipyretics for days, and studied the amount of nitrogen in the urine and feces. All the antipyretics except quinine increased the amount of change in nitrogenous tissues. He found that antifebrin, in doses of .08-.11 gr. per kilogramme of body-weight, exercised no marked influence; but in doses of .16-.19 grains caused an increased

action upon the decomposition of albumen tissues of 31-36 percent. The increased excretion of nitrogen on the antifebrin days was completely balanced by the decrease of nitrogen in the period after the stoppage of antifebrin. Antipyrin in large doses, 51 grammes in 16 days, caused no change in the albumen bearing tissues, although the uric acid was increased two-thirds beyond normal. Thallin, in doses of

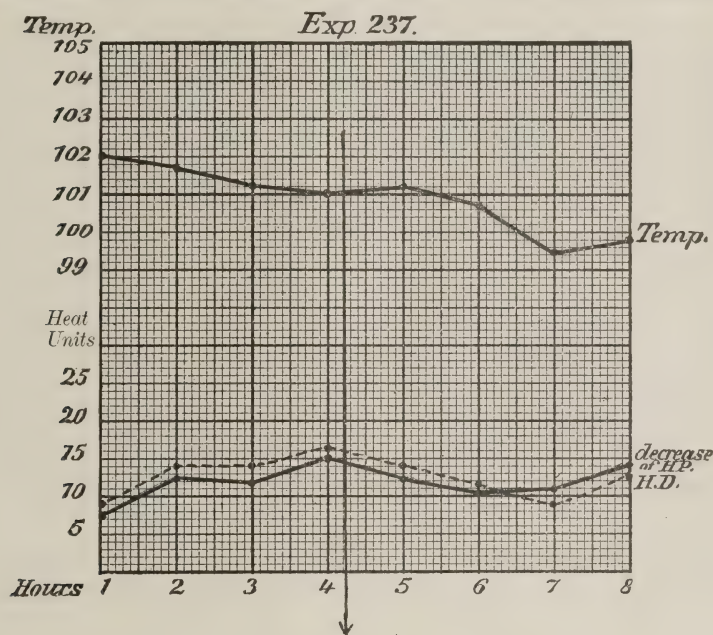


Fig. 8.

.014-.14 grains per kilogramme of body-weight, caused an increase of change in the albumen tissues (about seven per cent).

Quinine, in doses of .02-.04 grains per kilogramme, decreased the change in albumen tissues 9-16 per cent. and decreased the uric acid 13-50 per cent.

All the antipyretics except quinine increase the changes in albumen-tissues. Quinine decreases the albumen changes and the excretion of uric acid.

With antipyrin Prof. Chittenden found an increased excretion of urea, whilst it had a special inhibitory influence upon uric acid.

Now, with antipyrin and antifebrin, other experimentalists have found directly opposite results—some that metabolism is decreased, others that it is increased. Now, one factor, an important one to my mind, has not been taken into account, and that is the changes in external temperature. It is quite evident that on a cold day more heat is dissipated than on a warmer day, hence more metabolism

Exp. 229.

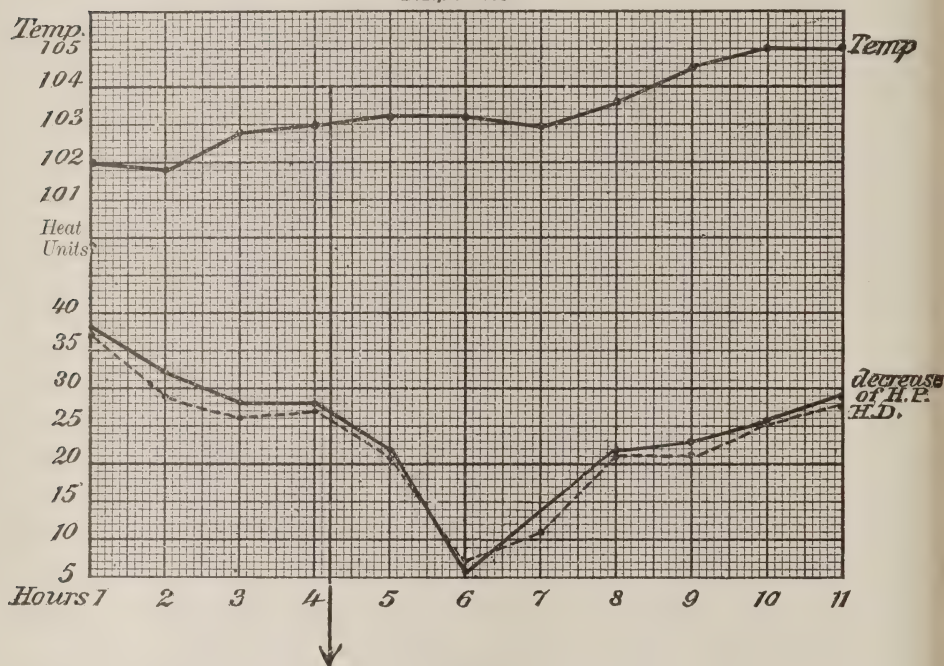


Fig. 9.

must ensue. Now, I believe that no results can be drawn of much accuracy unless the animals can be kept at the same external temperature. Further, I have shown that antipyrin acts on a thermotaxic centre whose function is polypnœa; and Sawadowski has seen no fall of temperature after removal of the corpus striatum from a dose of antipyrin.

All these researches lead me to believe that the antipyretics do not act on metabolism, but on the thermotaxic

centres, whose function it is to maintain the balance between heat production and heat dissipation, so that hyperpyrexia may not continue.

My calorimeter experiments with antipyrin gave the same antagonistic results, sometimes showing increased heat production, at other times decreased production, but usually immediately after the drug a temporary decrease of temperature, heat production, and heat dissipation.

I will now return to the circumscription of the basal thermotaxic centres, the only point upon which Dr. Girard and I differ. The centre between the thalamus and the corpus striatum and the centre at the anterior inner end of the thalami have been electrically irritated and found to be accompanied often by a rise of temperature. The caudate nucleus has also been electrically irritated and often attended by a rise of temperature. The rise of the temperature, when the parts between corpus striatum and optic thalamus have been mechanically insulted, is usually 105.5° F., which lasts three days; whilst the gray matter beneath the corpus striatum under similar treatment has a temperature of 105° F., which also lasts about three days. Now, punctures by a probe; a fine one may be made in the neighborhood of these centres, but the rise is not as great as at the points mentioned and falls greatly next days, as I have often seen, and which Girard's temperatures also prove. The thalamic thermotaxic centre is usually accompanied by a temperature curve totally different from the others; it agrees with these in being rapid in its ascent, but instead of lasting three days it lasts about three hours, and attains the colossal height of $109\frac{1}{2}^{\circ}$ F., and returns to normal in about six hours. Dr. Girard has never probed this centre, for he never attained in all his experiments a temperature over 106.8° about this centre. A puncture an eighth of an inch behind this centre will be followed often by a temperature of 105° , which remains sometimes till next day or falls rapidly back to normal. Now, the whole thermal apparatus is so very sensitive that a puncture anywhere in it may be accompanied by a temporary rise of temperature, and a puncture in the

neighborhood of a thermotaxic centre may so disorder it that it may be accompanied by a low rise of temperature, which may last till next day. I see no reason to doubt that the thalamic is not circumscribed. As to the caudate nucleus, it takes twenty-four to forty-eight hours for its lesion to attain a temperature of 107° , which is the highest temperature accompanying this injury.

No other thermotaxic centre in the brain is like it in that respect, all the other five being rapid risers. The rise of temperature after insults to caudate nucleus is accompanied by an augmented absorption of oxygen, excretion of carbonic acid and urea, according to Messrs. Sachs and Aronsohn. The great difference in these basal thermotaxic centres, with regard to their accompanying temperature curves as to rapid and slow rise of temperature, their short and long duration make it self-evident that we are dealing with different centres.

It has been thought that the fibres coming from the cortical thermotaxic centres might be injured and concerned in this rise of temperature about the basal thermotaxic centres; but the manner of the rise and the duration of the temperature excludes this view. For instance, the temperature curve after injury of caudate nucleus, and after lesion of the cruciate cortex centre above it, are totally different. Neither for the same reason can the temperature variations, by insults to centres behind or beneath it, be ascribed to the caudate nucleus. It makes a great difference whether you select the point where a lesion with probe is accompanied by the highest temperature, or to a point in the neighborhood where you have temperatures lower and not lasting as long as those made nearer the thermotaxic centre. All opinion is that the sensory fibres stand in a very close relation with these thermotaxic centres, in order to regulate the relation between H. P. and H. D. The relation of the sensory nerves to the polypnœic centre, which is a regulator of heat, also confirms this view.

If these centres were not circumscribed, then usually a probe should cause about the same rise of temperature all over the base of the brain, and this rise should continue

about the same length of time. But, as a fact, the probe causes temperature curves not equal in height or duration, and this I regard as a strong argument in favor of circumscribed centres. The real question is where do you usually obtain the highest temperature, and how wide is this area? The point at issue is not a mere rise of temperature of small amount and short in duration. The difficulty which Dr. Girard erects is that he has a series of small rises, about 105° F., in nearly all his observations upon the first day and less upon the second day; that there cannot be points where lesions are accompanied by temperatures much higher and this point be circumscribed. If a line be drawn from the most anterior thermotaxic centre to the most posterior of the basal centres, it will measure about half an inch; at one end by puncture a temperature attains its height, in twenty-four to forty-eight hours, of 107° , and lasts about three days. At the other end of the half inch, by puncture a temperature of $109\frac{1}{2}^{\circ}$ is attained in three hours, and lasts but a short time, returning to normal in four or five hours. Between these two points are two other centres which when punctured are associated with temperatures of 105° – 105.5 , rapidly reached and continued about three days. Temperature is as sensitive to lesion about these thermotaxic basal centres as arterial tensions to irritations of distant parts; but these changes in arterial pressure do not prove that no chief vaso-motor centre exists. I should like to state here that whilst a rise of temperature is often attained by insults to these centres at the base of the brain, it by no means follows that it always does; for I have often seen the reverse happen with the same sized probe, under the same external circumstances, and about in the same situation in the brain. I regard this fact an important proof that these centres preside over the relation of heat production to heat dissipation; and if this relation is disordered so that the temperature falls, it shows that the thermotaxic centres are not necessarily connected directly with heat production.

My colorimetric experiments upon the thermotaxic centres, when continued for several hours, showed sometimes increased production, at other times a decreased production of heat.

Usually there was a temporary elevation of heat production shortly after the injury. At the late Congress, Dr. Ferrier alluded to these thermotaxic centres as probably concerned in trophic work, and inferred that there must be several to preside over the tissues of the body. I have frequently seen great wasting of the body in large and well-developed rabbits after injury of the caudate nucleus, although their appetite was excellent. A similar wasting had been reported in the case of a boy with lesion of the corpus striatum.

The following are the conclusions from the experiments:

1. That the thermotaxic centre situated in the gray matter at the most anterior part of the third ventricle is the same as the thermo-polypnœic: polypnœa is a function of this thermotaxic centre.

2. That this centre acts reflexly, so that when heat is thrown on the body the sensory impulses excited by the heat are conveyed to this thermotaxic centre, which stirs up a thermolytic centre, the respiratory one to throw off heat. It stands between heat production on one side and heat dissipation on the other.

3. That the fall of the number of respirations by heat, after the removal of the above-mentioned centre, is due to an excitation of fibres running in the vagi which inhibit the respiratory centre.

4. That the normal temperature of the body is not necessarily dependent upon the amount of heat produced; for human calorimetry shows that the heat production varies, but the temperature remains nearly the same. The relation of heat production to heat dissipation decides the temperature which is regulated by the thermotaxic centres.

5. The cortical centres, the cruciate (Eulenberg and Landois) and the Sylvian are thermotaxic.

6. The four basal thermotaxic centres are situated as follows: one in the caudate nucleus (Sachs and Aronsohn), one in the gray matter beneath the corpus striatum, one in the gray matter about the most anterior part of the third

ventricle,, and still another in the gray matter at the anterior inner end of the optic thalamus.

7. That these six thermotaxic neither inhibit, except indirectly, nor excite heat production, but are thermotaxic, that is, regulating the relation of heat production to heat dissipation. That the thermolytic centres are the vaso-motor, respiratory, and sudorific ; that the thermolytic respiratory centre stands in relation to one of the basal thermotaxic centres ; that the thermolytic vaso-motor centre probably stands in part in relation to the thermotaxic centre, the caudate nucleus. The thermogenic centres are situated mainly in the spinal cord.

8. The thermotaxic centres are more circumscribed than Dr. Girard, of Geneva, believes.

9. That in fever neither increased production nor decreased dissipation, nor high temperature are necessary, but that fever is mainly a disease of thermotaxis, a disorder of the four basal thermotaxic centres. It is true that in septic fever, in its initial stage, heat production usually runs temporarily ahead of heat dissipation, but exceptionally both are immediately diminished.

10. That the antipyretics, as a rule, neither inhibit nor excite metabolism in a direct manner, but act upon the thermotaxic centres disordered by fever, to restore order or normal thermotaxis. Usually temporarily an antipyretic is accompanied by a diminished heat production and heat dissipation.

The number of experiments upon which this paper is founded is eighty-seven, part of which are given.

R. means number of respirations.

R. T. " rectal temperature.

C. T. " calorimeter temperature.

A. T. " air temperature.

E. T. " exit tube temperature (Centigrade).

M. " meter, amount in litres.

B. T. " temperature of hot box.

Lab. T. " temperature of laboratory.

H. D. " heat dissipation.

H. P. " heat production.

When the thermotaxic centres were punctured, I used a probe two millimetres in width and one in thickness. The tube used in operation upon the basal thermotaxic centres was two millimetres in diameter.

EXP. 165.—Rabbit. Cortex partly destroyed.

P. M.	RESP.	R. T.	B. T.	L. T.
2.12	20	103.5	100	74
2.18	37	103.5		
2.25	45	103.5		
2.35	58	104.2		
2.54	66	105.8	102	
3.15	43	107.8		
3.27	43	109.7	102	
3.35	30	110.4		

EXP. 166.—Rabbit. Cortex destroyed.

TIME, P. M.		RESP.	R. T.	B. T.	LAB. T.
1.42	Put in warm box.	49	102.6	104	80
1.50		73	103.	100	
2.4		78	104.	100	
2.20		94	106.1	100	
2.35		103	107.1	102	
2.46		81	108.4	104	
2.52		84	108.4	100	
3.7		50	109.2	102	
3.16		41	109.2	100	
3.27		38	110.2	104	

EXP. 168.—Rabbit. Cortex removed.

P. M.	RESP.
1.50.....	26
1.55 Electrodes upon the space between corpora striata and optic thalami.	
1.57.....	42
2.32 Electrodes as above..	27
2.34.....	54

EXP. 175.—Rabbit. Cortex removed.

P. M.	RESP.
2.21 Electrodes as in Exp. 168.	20
2.24.....	54
2.30.....	20

EXP. 170.—Rabbit. Cortex and corpora striata removed, also the parts at anterior end of 3d ventricle.

P. M.	RESP.	R. T.	B. T.	LAB. T.
2.10	14	99.4	101	76
2.17	11	99.0	101	
2.37	11	100.	97	
2.48	15	102.	102	
3.7	22	102.3	104	
3.20	28	104.4	106	
3.33	21	106.4	104	
3.39	20	109.1		
4.7	Died.	110.8		

EXP. 171.—Rabbit. Cortex removed and also corpora striata, with parts between them and optic thalami.

P. M.		RESP.	R. T.	B. T.	LAB. T.
1.55	Put in box.	17	103.2	110	84
2.		15	103.4	105	
2.5		15	104.3	100	
2.18		15	104.3	106	
2.27		18	105.2	106	
2.37		17	106.1	106	
2.47		23	107.0	102	
3.2		26	108.1	106	
3.18		24	109.2	100	
3.33		25	110.2	104	

EXP. 162.—Rabbit. Polypnœic centre removed.

P. M.	RESP.	R. T.	B. T.	LAB. T.
2.00	31	101.4	102	78
2.10	20	102.	100	
2.30	18	103.8	100	
2.45	30	105.2	100	
3.0	28	107.4	100	
3.15	29	108.8	100	
3.30	34	110.4	102	
3.40	33	111.8	100	
3.55	Dying.	113.		

EXP. 172.—Rabbit. Cortex destroyed, polypnœic centre destroyed on both sides.

P. M.	RESP.	R. T.	B. T.	LAB. T.
1.50	14	102.2	105	78
1.51	12	102.	100	
2.10	14	102.4	98	
2.20	13	104.	102	
2.37	14	106.	100	
2.53	17	108.1	102	
3.8	17	110.1	104	
3.12	17	110.3	104	

EXP. 177.—Rabbit. Cortex removed; a puncture made into the anterior end of the gray matter about the 3d ventricle.

P. M.		RESP.	R. T.	B. T.	LAB. T.
1.35	Put in box.	13	103 $\frac{3}{5}$	100	26
1.38		12	103 $\frac{1}{5}$	100	
1.42		19	103 $\frac{1}{5}$		
1.51		19			
2.8		15	104.0	104	
2.22		16	105.2	106	
2.40		31	107.1	108	
2.42		19	107	108	
2.50		19	107	108	
3.6		19	108	108	

Autopsy: polypnœic centre partly destroyed.

EXP. 181.—Rabbit. Cortex removed, corpora striata only destroyed.

P. M.	RESP.	R. T.	B. T.
3.30	17	104 $\frac{1}{5}$	100
3.33	36		
3.38	66	107 $\frac{1}{5}$	116

EXP. 182.—A section made between pons and medulla oblongata at lower border of pons.

2.1	25	104	108	78
2.5	31	104 $\frac{1}{5}$	108	
2.15	33	105.1	110	
2.32	30	108	112	
2.42	32	109	110	
3.5 Dying.	33	111 $\frac{3}{5}$	110	

EXP. 183.—Rabbit. Transverse section through the crura cerebri, leaving corpora quadrigemina intact.

P. M.		RESP.	R. T.	B. T.	LAB. T.
3.27	Put in box.	22	105 $\frac{2}{5}$		80
3.32		19	105 $\frac{4}{5}$	108	
3.48		19	106.1	104	
4.1		22	107	100	
4.26		26	109.1	104	
4.55		28	111	106	

EXP. 184.—Rabbit. Transverse section through the pons; convulsions in left posterior leg.

P. M.		RESP.	R. T.	B. T.	LAB. T.
1.31		15	103 $\frac{2}{5}$		72
1.32		14		94	
1.37		11	103 $\frac{2}{5}$	94	
1.52		12	101 $\frac{2}{5}$	96	
2.10		15	107	100	
2.20	Dying; respiratory movements, all inspiratory		108 $\frac{1}{5}$	102	

EXP. 186 —Rabbit. Section just behind corpora quadrigemina, leaving pons and medulla intact.

P. M.	RESP.	R. T.	B. T.	LAB. T.
2.35	18	102 $\frac{3}{5}$	98	68
2.43	21	101 $\frac{4}{5}$	98	
2.54	17	103 $\frac{3}{5}$	98	
3.6	16	105 $\frac{1}{5}$	100	
3.17	15	107	102	
3.26	16	107 $\frac{4}{5}$	104	
3.35	22	109 $\frac{1}{10}$	100	

EXP. 188.—Rabbit. Section transversely at anterior edge of corpora quadrigemina down to the base of brain, carotids previously ligated.

P. M.	RESP.	R. T.	B. T.	LAB. T.
2.	14	100 $\frac{1}{5}$	104	64
2.5	14	100 $\frac{1}{5}$	102	
2.15	16	100 $\frac{5}{5}$	102	
2.24	16	101 $\frac{4}{5}$	102	
3.16	18	108 $\frac{2}{5}$	106	
3.22	22	109.1	106	

EXP. 192.—Rabbit. Carotids ligated, trigemini divided, transverse section just behind the polypnœic centre.

P. M.	RESP.	R. T.	B. T.	LAB. T.
4.7	25	102 $\frac{3}{5}$	100	72
4.10	Put in box.			
4.17	18	102 $\frac{4}{5}$	102	
4.25	17	103 $\frac{2}{5}$	100	
4.33	14	104	100	
4.47	15	105	100	
5.13	16	109	112	
5.26	20	109 $\frac{4}{5}$	98	

EXP. 195.—Rabbit. Vagi cut, cortex destroyed, polypnœic centre cut off by a transverse section at anterior part of optic thalami.

P. M.	RESP.	R. T.	B. T.	LAB. T.
1.15	12	101 $\frac{1}{5}$	92	70
1.22	12	102 $\frac{2}{5}$	98	
1.30	12	101 $\frac{4}{5}$	102	
1.49	12	102 $\frac{3}{5}$	102	
2.1	12	103	100	
2.34	12	106 $\frac{4}{5}$	98	
3.12	16	110 $\frac{2}{5}$	100	

EXP. 196.—Rabbit. Cortex, vagi cut; polypnœic centre removed by a transverse section.

P. M.	RESP.	R. T.	B. T.	LAB. T.
3.40	16	104 $\frac{4}{5}$	100	66
3.45	15 $\frac{1}{2}$		98	
3.52	16 $\frac{1}{2}$	104 $\frac{1}{5}$	98	
4.1	19	105 $\frac{3}{5}$	98	
4.14	23	106 $\frac{4}{5}$	98	
4.30	22	109 $\frac{1}{5}$	98	

EXP. 238.—Rabbit.

P. M.	R. T.
12.20.....	102.2
12.30 Transverse section through the middle of the pons.	
1.10	99 $\frac{3}{5}$
1.40.....	99.6
2.5	99.4
2.34.....	99.8
3.5	100.6
3.47.....	101.2

EXP. 239.—Rabbit.

P. M.	R.	R. T.	BOX T.
1.55	10 grs. of antipyrin subcutaneously..	17	103.3
3.		12	101.3
3.17		25	101.8
3.35		31	103.4
3.55		31	105.6
4.10		37	108.1
4.30	Died.		111

EXP. 240.—Rabbit.

P. M.	R.	R. T.	BOX T.
1.40	10 grs. of antipyrin subcutaneously.		
2.20	5 grs. of antipyrin subcutaneously.		
2.3	120	101.1	86
2.4	150	102	86
3.	28	103.2	92
3.15	19	104.6	98
3.35	20	107.7	106
3.45	21	109.8	104
	Died.		

EXP. 242.—Rabbit.

P. M.	R. T.
12.34.....	100.4
1.40	Transverse section of brain at posterior end of optic thalami.
2.5	101.5
3.12.....	100.5
4.20.....	99.2

EXP. 246.—Rabbit; heated up.

P. M.	R.	R. T.
2.	95	103.4
2.15	8 grains of antipyrin by the jugular. The animal is removed from the heated box and lies on the table.	
2.16		98
2.17.....		92
2.18.....		93
2.19.		96
2.22		92
2.23.....		82
2.24.....		69
2.26... ..		62

2.39.....	32
2.31.	33
2.32	21
2.33.....	11
2.42.....	6
2.47.....	7

EXP. 243.—Rabbit.

P. M.	R.	R. T.	B. T.
1.30	4 gtts. of blood subcutaneously.		
3.55	7	102.5	102
4.	7	102.5	
4.12	17	102.9	
4.20	34	103.8	104
4.30	49	104.4	
4.40	57	106.	
4.55	58	106.5	
5.	56	108.	
5.10	36	109.	
5.15	30	110	
5.25	29	110.4	108

EXP. 245.—Rabbit.

P. M.	R.	R. T.
3.	24	103.7
3.29.45	85	106.
3.30	Dropped into ice water and kept in	
	23	
3.30.15	40	
3.30.30	44	
3.30.45	48	
3.31.45	56	
3.32.45	71	
3.35.0	31	

EXP. 244.—Rabbit. Hair closely clipped over the whole body.

	R.	R. T.	B. T.
12.00 M.		102.2	
P. M.			
12.10	Covered with mucilage.		
1.5	7	98.6	100
1.12	7	97.2	
1.21	7	96.6	96
1.33	7	96.6	102
1.43	6	96.8	104
2.10	7	98.8	100
3.15	8	101.2	102
3.30	60	102.2	108
3.45	78	102.8	104
4.	53	105.2	
4.23	30	108.	

EXP. 202.—Rabbit. Carotids tied, cortex lifted up, thalamic bared, and a hollow tube two millimetres in diameter pushed down into the anterior ends of both thalami.

P. M.	R. T.
3.45	102 $\frac{4}{5}$
3.46 Operation performed.	
3.56	102 $\frac{4}{5}$
4.17	102
4.45	102.1
5.	102.1
6.	103 $\frac{3}{5}$
7.30	104 $\frac{4}{5}$
9.30	104 $\frac{4}{5}$
<i>2d day.</i>	
8 A. M.	101 $\frac{2}{5}$

EXP. 203.—Rabbit. Cortex lifted up, anterior end of right thalamus tubed, carotids tied, runs to left.

P. M.	R. T.
2.	103 $\frac{4}{5}$
2.10 Operation performed.	
2.40	103 $\frac{3}{5}$
3.3	103 $\frac{4}{5}$
3.30	103 $\frac{1}{5}$
3.44	103 $\frac{3}{5}$
4.29	103 $\frac{3}{5}$
4.58	103 $\frac{3}{5}$
6.45	103 $\frac{2}{5}$
9.30	102
<i>2d day.</i>	
8 A. M.	93.2

EXP. 204.—Rabbit.

P. M.	R. T.
1.20	102
1.30 Right optic thalamus punctured about its middle.	
2.7	101 $\frac{3}{5}$
2.40	101 $\frac{4}{5}$

P. M.	R. T.
3.15.....	102 $\frac{1}{5}$
3.30.....	101 $\frac{9}{10}$
4.....	102 $\frac{2}{5}$
4.30.....	102 $\frac{1}{5}$
5.....	102 $\frac{1}{5}$
6.....	103
8.....	101 $\frac{2}{5}$

EXP. 205.—Rabbit. Cortex lifted up.

P. M.	R. T.
1.45.....	103 $\frac{3}{5}$
1.50 Right optic thalamus tubed about its middle.	
2.17.....	102 $\frac{1}{5}$
2.44.....	101 $\frac{1}{5}$
3.18.....	101
3.35.....	100 $\frac{4}{5}$
4.....	101 $\frac{3}{5}$
4.30.....	101 $\frac{3}{5}$
5.....	101 $\frac{2}{5}$
6.....	101 $\frac{3}{5}$
8.....	98 $\frac{2}{5}$

EXP. 206.—Rabbit. Carotids tied, cortex lifted.

P. M.	R. T.
1.30.....	103
1.35 Optic thalamus punctured about its middle.	
1.57.....	101 $\frac{1}{5}$
2.35.....	102 $\frac{1}{5}$
3.3.....	101 $\frac{3}{5}$
3.20.....	101.2
3.39.....	101 $\frac{2}{5}$
4.14.....	101 $\frac{2}{5}$
Ligature removed from the carotids.	
4.30.....	100 $\frac{9}{10}$
4.445.....	100 $\frac{1}{5}$
5.45.....	101
8.....	102
9.....	100.9

EXP. 207.—Rabbit. Carotids tied.

P. M.	R. T.
2.20.....	103 $\frac{3}{5}$
2.30 Right optic thalamus tubed about its middle, cortex lifted up.	
2.58.....	102
3.14.....	102 $\frac{1}{5}$
3.35.....	102 $\frac{3}{5}$
3.59.....	103 $\frac{1}{5}$
4.45.....	103 $\frac{1}{5}$
5.	102 $\frac{4}{5}$
5.40.....	101 $\frac{3}{5}$
8.	101

EXP. 208.—Rabbit.

P. M.	R. T.
4.25.....	103
4.30 Thalamic, thermotaxic centre had a tube two millimetres through it.	
4.45.....	102 $\frac{1}{5}$
5.	102 $\frac{2}{5}$
5.15.....	102 $\frac{1}{5}$
5.37.....	102 $\frac{1}{5}$
6.45.....	102 $\frac{4}{5}$
8.	103
9.30.....	103 $\frac{4}{5}$

EXP. 209.—Rabbit.

P. M.	R. T.
1.30.....	103
1.35 Thalamic thermotaxic centre destroyed.	
2.8.....	101 $\frac{1}{5}$
2.32.....	101
2.56.....	101
3.21.....	101 $\frac{1}{5}$
3.40.....	101 $\frac{4}{5}$
3.59.....	101 $\frac{4}{5}$
4.20.....	102
4.40.....	102 $\frac{3}{5}$
5.45.....	103 $\frac{2}{5}$
6.45.....	104 $\frac{2}{5}$
8.40.....	101 $\frac{4}{5}$

2d day.

8 A. M. 103

EXP. 210.—Rabbit.

P. M. R. T.

1.40 103 $\frac{1}{5}$ 1.45 Thermotaxic centre at anterior inner end
of gray matter of 3d ventricle tubed in
part; respirations 240 per minute;
thermo-polypnœa.2.13 100 $\frac{4}{5}$ 2.36 100 $\frac{3}{5}$ 3.1 100 $\frac{2}{5}$ 3.32 100 $\frac{4}{5}$ 3.50 99 $\frac{3}{5}$

4.08 99.9

4.27 99.8

4.45 99.8

5.50 99 $\frac{3}{5}$

6.50 100

8.40 101 $\frac{1}{5}$ *2d day.*8 A. M. 102 $\frac{5}{8}$ *EXP. 211.*—Rabbit.

P. M. R. T.

11.20 101.1

11.25 The gray matter of the 3d ventricle at its
most anterior part had a tube two milli-
metres in diameter driven through it;
corpus striatum slightly injured.11.30 99 $\frac{3}{5}$ 11.45 99 $\frac{3}{5}$

12.27 101

1.12 100 $\frac{1}{5}$ 1.33 99 $\frac{2}{5}$ 1.59 99 $\frac{1}{5}$ 2.32 98 $\frac{4}{5}$ 2.53 98 $\frac{4}{5}$ 3.34 98 $\frac{3}{5}$

6.10 100

9.20 101 $\frac{1}{5}$

*2d day.*8 A. M. 101 $\frac{1}{5}$ *EXP. 212.*—Rabbit.

P. M.	R. T.
11.35	103
11.40 The gray matter of the 3d ventricle at its most anterior part had a tube pressed through it; corpus striatum partly in- jured.	
11.54	100
11.35	102
1.15	103 $\frac{4}{5}$
1.37	104 $\frac{1}{5}$
2.05	104 $\frac{2}{5}$
2.37	103 $\frac{3}{5}$
2.57	101 $\frac{1}{5}$
4.35	102 $\frac{3}{5}$
9.20	104 $\frac{3}{5}$
8 A. M., 2d day—convulsions.	98

EXP. 213.—Rabbit.

P. M.	R. T.
12.40	103
12.50 Caudate nucleus cleanly sucked out.	
1.20	102 $\frac{2}{5}$
1.40	102 $\frac{2}{5}$
2.10	102 $\frac{3}{5}$
3.18	101 $\frac{4}{5}$
3.41	102 $\frac{1}{5}$
4.39	102 $\frac{3}{5}$
6.20	102.9
9.30	102.2
8 A. M., 2d day	103

EXP. 214.—Rabbit.

P. M.	R. T.
1.	103 $\frac{3}{5}$
1.10 Tissue beneath corpus striatum broken up.	
1.22	102

P. M.	R. T.
1.44	102
2.13	101 $\frac{4}{5}$
2.45	101 $\frac{4}{5}$
3.7	101 $\frac{2}{5}$
3.22	101 $\frac{1}{5}$
4.35	102
6.25	102.9
9.20	103 $\frac{3}{5}$
8 A. M., 2d day.	103 $\frac{2}{5}$

EXP. 216.—Rabbit; fasted 3 days, allowed water; wt. 3.86 lbs.

TIME.	A. T.	C. T.	E. T.	R. T.	METER.
7.30	66.6	66.6	18 $\frac{2}{5}$	101.1	95,544 litres.
8.30	68.5	67.0	19	101.8	96,385 "

4

— .3

H. D. = 16.68. H. P. = 15.82. Wt. 3.74 lbs.

8.35	69.1	67.	19	101.8	96,385 "
9.35	70.	67.4	19 $\frac{1}{5}$	102.4	96,995 "

.4

.6

H. D. = 16.68. H. P. = 18.54. Wt. 3.74 lbs.

9.41	70.	67.4	19 $\frac{1}{5}$	102.4	96,995 "
10.41	70.1	67.8	19 $\frac{3}{5}$	101.8	97,365 "

.4

— .6

H. D. = 16.68. H. P. = 14.84. Wt. 3.70 lbs.

10.52	70.1	67.8	19 $\frac{3}{5}$	101.8	97,365 "
11.52	71.1	68.18	19 $\frac{1}{5}$	101.8	97,772.5 "

.0

12 M. Puncture by a tube two millimetres through the gray matter at the most anterior end of the 3d ventricle.

12.17	72.6	68.18	20	101.	97,725 litres.
12.17	72.0	68.4	20	102.4	98,295 "

+ .22

+ 1.4

H. D. = 9.178. H. P. = 13.46. Wt. 3.70 lbs.

1.21	72.	68.4	20	102.4	98,295 litres.
2.21	73.1	68.75	20	103.8	98,610 "

 $+ .35$ 1.4

H. P. = 14.60. H. P. = 18.89. Wt. 3.70 lbs.

2.25	73.1	68.75	20.	103.8	98,610
3.25	74.5	69.2	20.4	104.6	99,050

 $+ .45$

H. D. = 18.77. H. P. = 21.21. Wt. 3.68 lbs.

3.30	74.5	69.2	20.4	104.6	99,05C	'
4.30	74.5	69.5	20.6	104.6	99,710	'

· 3

H. D. = 12.51. H. P. = 13.12. Wt. 3.68 lbs.

4.35	74.5	69.5	$20\frac{3}{5}$	$104\frac{3}{5}$	99,710
5.35	71.8	69.9	$20\frac{4}{5}$	$104\frac{3}{5}$	101,019

.4

H. D. = 16.68. H. P. = 17.29. Wt. 3.68 lbs.

5.42	71.8	69.9	$20\frac{4}{5}$	$104\frac{3}{5}$	101,019	“
6.42	71.4	70.3	$21\frac{1}{5}$	$104\frac{4}{5}$	102,218	“

.4

 $+\frac{1}{5}$

H. D. = 16.68. H. P. = 17.29.

EXP. 217.—Rabbit; fasted 3 days.

A. M.	A. T.	C. T.	E. T.	R. T.	LITRES.
7.17	70.7	70.7	21.2	101.1	
8.17	71.4	71.4	21.4	101.8	1,122

.7

H. D. = 29.20. H. P. = 18.25. Wt. 3.82 lbs.

8.20	71.4	71.4	21.4	101.8	
9.20	76.1	71.9	21.8	101.8	945

5

H. D. = 10.86. H. P. = 20.86. Wt. 3.82 lbs.

9.25	76.1	71.9	21.8	101.8	
10.25	73.6	72.4	22.	102.2	1,015

• 5

$+ .4$

H. D. = 20.86. H. P. = 24.06. Wt. 3.82 lbs.

10.32	73.6	72.4	22.	102.2	
11.32	74.8	72.9	22.4	101.9	1,015
<hr/>					
.5				.3	

H. D. = 20.86. H. P. = 19.91.

11.40 A. M. A tube two millimetres in diameter was pushed through the anterior inner end of optic thalamus.

Wt. 3.82 lbs.

12.	75.	72.9	22.6	101.2	
1.	76.3	73.4	22.4	99.6	1,055

H. D. = 20.86. H. P. = 15.79. Wt. 3.78 lbs.

1.7	76.3	73.4	22.4	99.6	
2.7	73.1	73.8	22.8	99.8	1,230
<hr/>					
.4				+ .2	

H. D. = 16.68. H. P. = 17.31. Wt. 3.78 lbs.

2.15	74.1	73.8	22.8	99.8	
3.15	75.2	74.2	23.0	101.2	1,060

1.4

H. D. = 16.68. H. P. = 17.20. Wt. 3.76 lbs.

3.22	75.2	74.2	23.	101.2	
4.22	77.3	74.8	23.4	102.4	1,080

H. D. = 25.03. H. P. = 28.75. Wt. 3.74 lbs.

4.25	75.3	74.8	23.4	102.4	
5.25	77.7	75.35	23.8	104.1	1,034

.55

+ 1.9

H. D. = 22.94. H. P. = 28.83.

EXP. 218.—Rabbit; fasted 2½ days; wt. 4.62 lbs.

A. M.	A. T.	C. T.	E. T.	R. T.	LITRES.
7.16	72.2	70.5	20.2	102.0	
8.16	73.7	70.90	20.4	101.8	1,141

H. D. = 16.68. H. P. = 15.92. Wt. 4.62 lbs.

8.24	73.7	70.9	20.4	101.8	
9.24	73.6	71.15	20.8	102.2	985

.25

.4

H. D. = 10.43. H. P. = 11.19. Wt. 4.62 lbs.

9.29	73.6	71.15	20.8	102.2	
10.29	73.9	71.4	21.2	102.6	945
		<hr/>		<hr/>	
		.25		+.4	

H. D. = 10.43. H. P. = 11.19. Wt. 4.62 lbs.

10.38	73.9	71.4	21.2	102.6	
11.38	74.8	71.88	21.4	102.3	950
		<hr/>		<hr/>	
		.48		-.3	

H. D. = 20.02. H. P. = 17.82.

12 M. Caudate nucleus in greater part sucked out.

Wt. 4.52 lbs.

12.5	75.3	71.7	21.4	101.4	
1.5	76.05	72.1	21.8	102.6	1,116
		<hr/>		<hr/>	
		.4		1.2	

H. D. = 16.68. H. P. = 21.18. Wt. 4.48 lbs.

1.15	76.05	72.1	21.8	102.2	
2.15	74.8	72.5	22.2	103.1	2,454
		<hr/>		<hr/>	
		.4		+.9	

H. D. = 16.68. H. P. = 20.02. Wt. 4.46 lbs.

2.20	74.8	72.5	22.2	103.1	
3.20	75.6	73.1	22.2	102.8	2,730
		<hr/>		<hr/>	
		.6		-.3	

H. D. = 24.03. H. P. = 25.24. Wt. 4.46 lbs.

3.25	75.6	73.1	22.2	102.8	
4.25	74.5	73.5	22.2	102.6	2,690
		<hr/>		<hr/>	
		.4		.2	

H. D. = 16.68. H. P. = 15.94.

4.30	74.5	73.5	22.2	102.6	
5.30	77.	74.	22.8	102.4	2,690
		<hr/>		<hr/>	
		.5		-.2	

H. D. = 20.86. H. P. = 20.12.

EXP. 219.—Rabbit; fasted $2\frac{1}{2}$ days; wt. 3.60 lbs.

A. M.	A. T.	C. T.	E. T.	R. T.	LITRES.
7.15	71.7	71.2	21.0	102.	
8.15	74.2	71.8	21.3	100.8	1,210

.6

— 1.2

H. D. = 25.03. H. P. = 21.45. Wt. 3.56 lbs.

8.25	74.1	71.8	21.3	100.8	
9.25	73.5	72.15	21.4	100.2	1,040

.35

— .6

H. D. = 14.60. H. P. = 12.83. Wt. 3.56 lbs.

9.34	73.5	72.15	21.4	100.2	
10.34	74.	72.5	21.4	100.4	1,365

.2

H. D. = 14.60. H. P. = 15.19.

10.40	74.	72.5	21.4	100.4	
11.40	74.6	72.9	22.2	100.4	1,095

.4

H. D. = 16.68. H. P. = 16.68.

11.45. A blunt probe thrust through the gray matter about the most anterior end of the 3d ventricle.

Wt. 3.42 lbs.

12.	74.6	72.9	22.2	99.6	
1.	73.6	73.0	22.2	101.	1,100

.1

1.4

H. D. = 4.17. H. P. = 8.14. Wt. 3.40 lbs.

1.3	73.6	73.0	22.2	101.0	
2.3	75.9	73.35	22.4	101.6	1,046

.35

+ .6

H. D. = 14.60. H. P. = 16.29. Wt. 3.36 lbs.

2.6	75.9	73.35	22.4	101.6	
3.6	75.4	73.7	22.6	101.8	1,145

.35

.2

H. D. = 14.60. H. P. = 15.15. Wt. 3.34 lbs.

3.11	75.4	73.7	22.6	101.8	
4.11	76.0	74.1	22.8	102.	950
		<hr/>		<hr/>	
		.4		+ .2	
H. D. = 16.68. H. P. = 17.23. Wt. 3.32 lbs.					

4.14	76.3	74.1	22.8	102.	
5.14	75.3	74.4	23.0	102.4	705
		<hr/>		<hr/>	
		.3		+ .4	
H. D. = 12.51. H. P. = 13.51.					

5.18	75.3	74.4	23.	101.6	
6.18	75.5	74.8	23.2	102.0	572
		<hr/>		<hr/>	
		.4		+ .4	
H. D. = 16.68. H. P. = 17.78.					

EXP. 220.—Rabbit; fasting 3 days; wt. 4.24 lbs.

A. M.	A. T.	C. T.	E. T.	R. T.	LITRES.
7.54	70.45	70.45	20.	101.2	
8.54	72.7	70.95	20.3	100.6	1,214
		<hr/>		<hr/>	
		.5		— .4	
H. D. = 20.86. H. P. = 19.46. Wt. 4 lbs.					
9.2	72.7	71.95	20.6	100.6	
10.2	72.1	72.2	20.8	100.6	2,140
		<hr/>		<hr/>	
		.25		.0	

H. D. = 10.43. H. P. = 10.43. Wt. 4 lbs.

10.10	72.1	71.2	20.8	100.6	
11.10	74.3	71.5	21.2	100.4	1,555
		<hr/>		<hr/>	
		.3		— .2	
11.15	74.3	71.5	21.2	100.4	
12.15	75.7	72.0	21.4	100.4	1,210
		<hr/>			
		.5			
H. D. = 20.86. H. P. = 20.86.					

12.20 Corpus striatum destroyed.

Wt. 3.98 lbs.

12.30	74.7	71.95	21.4	99.4	
1.30	72.7	72.1	21.7	100.	500
		<hr/>		<hr/>	
		.15		+.6	

H. D. = 6.25. H. P. = 6.45. Wt. 3.98 lbs.

1.35	72.7	72.1	21.7	100.	
2.35	77.0	71.35	21.8	100.4	1,515
		<hr/>		<hr/>	
		+.25		+.4	

H. D. = 10.43. H. P. = 11.75. Wt. 4.98 lbs.

2.40	75.5	72.35	21.8	100.4	
3.40	76.8	72.65	22.0	100.4	127
		<hr/>		<hr/>	
		.30		.0	

H. D. = 12.51. H. P. = 12.51. Wt. 3.96 lbs.

3.45	76.8	72.65	22.	100.4	
4.45	76.7	83.	22.2	100.4	1,463
		<hr/>		<hr/>	
		.35			

H. D. = 14.60. H. P. = 14.60. Wt. 3.96 lbs.

4.49	76.7	73.0	22.1	100.4	
5.49	74.55	73.3	22.6	100.8	846
		<hr/>		<hr/>	
		.3		+.4	

H. D. = 12.51. H. P. = 13.83.

EXP. 221.—Rabbit; fasted 3 days.

A. M.	A. T.	C. T.	E. T.	R. T.	LITRES.
7.20	74.0	67.9	20.5	102.2	
8.20	72.1	68.75	20.4	102.2	1,119
		<hr/>		<hr/>	
		.85			

H. D. = 35.45. H. P. = 35.45. Wt. 3.84 lbs.

8.27	72.1	68.75	20.4	102.2	
9.27	71.3	69.55	20.8	101.8	1,215
		<hr/>		<hr/>	
		.80		— .4	

H. D. = 33.37. H. P. = 32.10. Wt. 3.84 lbs.

9.34	71.3	69.55	20.8	101.8	
10.34	72.	70.1	21.2	101.9	1,213

+ .1

H. D. = 22.94. H. P. = 23.25. Wt. 3.84 lbs.

10.43	72.	70.1	21.1	101.8	
11.43	73.6	70.7	21.4	102.1	1,187

.6

.7

H. D. = 25.06. H. P. = 27.29.

11.50. Gray matter beneath corpus striatum destroyed with probe.

Wt. 3.83 lbs.

12.10	73.4	70.7	21.6	101.2	
1.10	71.6	71.1	21.6	101.6	1,215

.4

+ .4

H. D. = 16.68. H. P. = 17.95. Wt. 3.76 lbs.

1.14	71.6	71.15	21.6	101.6	
2.14	73.2	71.70	21.8	101.2	1,300

.55

— .4

H. D. = 22.94. H. P. = 21.70. Wt. 3.76 lbs.

2.19	73.2	61.70	21.7	101.2	
3.19	72.2	72.1	22.	100.8	2,755

.40

— .4

H. D. = 16.68. H. P. = 15.44. Wt. 4.64 lbs.

3.20	73.0	72.1	22.	100.6	
4.20	75.5	72.5	22.4	101.2	1,095

.4

.6

H. D. = 16.68. H. P. = 18.99. Wt. 3.64 lbs.

4.36	75.5	72.5	22.4	101.2	
5.26	76.9	72.9	22.7	101.4	1,795

+ .2

H. D. = 16.68. H. P. = 17.30.

EXP. 224.—Rabbit; fasted 2 days; wt. 4 28 lbs.

7.21	72.	71.9	21.2	102.0	
8.21	77.6	72.4	21.8	102.0	979

.5 .0

H. D. = 20.86. H. P. = 20.86. Wt. 4.28 lbs.

8.24	77.8	72.4	21.8	102.0	
9.24	75.2	72.8	22.2	102.0	1,082

.4

H. D. = 16.86. H. P. = 16.86. Wt. 4.28 lbs.

9.30	75.2	72.8	22.7	102.0	
10.30	65.4	73.2	22.4	102.0	1,348

.4

H. D. = 16.68. H. P. = 16.68. Wt. 4.26 lbs.

10.34	75.4	73.2	22.4	102.0	
11.34	76.4	73.9	22.6	101.6	2,525

.6

.4

H. D. = 25.03. H. P. = 23.62. Wt. 4.24 lbs.

11.40	76.4	73.8	22.9	101.6	
12.40	76.2	74.1	22.8	102.0	1,715

.3

+.4

H. D. = 12.51. H. P. = 13.90.

1.20 Puncture into thalamus at its anterior inner end.

2.7	76.0	73.9	22.8	102.4	
3.7	76.5	74.3	23.0	102.8	1,055

.4

+.4

H. D. = 16.68. H. P. = 18.08. Wt. 4.20 lbs.

3.12	76.5	74.3	23.0	101.8	
4.12	75.0	74.7	23.2	102.9	940

.4

.0

H. D. = 16.68. H. P. = 16.68. Wt. 4.20 lbs.

4.15	75.0	74.7	23.2	102.8	
5.15	77.2	75.1	23.4	103.0	1,065

.4

+.2

Wt. 4.20 lbs.

5.19	77.2	75.1	23.2	103.0	
6.19	76.	75.5	23.4	102.6	1, 24
		<hr/>		<hr/>	
		.3		— .4	
H. D. = 12.51. H. P. = 11.12.					

EXP. 224.—Rabbit; fasted 12 hours; wt. 4.62 lbs.

A. M.	A. T.	C. T.	E. T.	R. T.	LITRES.
7.19	71.4	71.4	21.	103.4	
8.19	73.1	70.3	21.6	103.4	1, 111
H. D. = 37.54. H. P. = 37.54. Wt. 4.50 lbs.					

8.23	73.1	72.3	21.4	103.4	
9.23	76.8	72.9	22.0	103.2	1, 055
		<hr/>		<hr/>	
		.6		— .2	
H. D. = 25.03. H. P. = 24.29. Wt. 4.48 lbs.					

9.28	76.8	72.9	22.0	103.2	
10.28	76.6	73.4	22.4	102.6	890
		<hr/>		<hr/>	
		.5		— .6	
H. D. = 20.86. H. P. = 18.63.					

10.35	76.6	73.4	22.4	102.6	
11.35	75.5	73.65	22.4	104.2	1, 130
		<hr/>		<hr/>	
		+ .25		+ 1.6	
H. D. = 10.43. H. P. = 17.56.					

11.45. Puncture into the gray matter at the most anterior end of the 3d ventricle.

12.				103.8	
12.10				104.1	
12.29	77.4	73.45	22.4	103.6	
1.29	65.2	74.2	22.8	103.4	995
		<hr/>		<hr/>	
		.75		— .2	
H. D. = 31.29. H. P. = 30.56. Wt. 4.44 lbs.					

1.35	76.2	74.2	22.2	103.4	
2.35	75.8	74.7	23.0	103.6	645
		<hr/>		<hr/>	
		.5		+ .2	
H. D. = 20.86. H. P. = 20.50. Wt. 4.40 lbs.					

2.44	75.8	74.7	33.0	103.6	
3.44	75.2	75.05	23.2	105.6	600

+ 35 2.0

H. D. = 14.60. H. P. = 22.00. Wt. 4.30 lbs.

3.49	75.2	75.05	23.2	105.3	
4.49	77.2	75.55	23.8	105.8	225

.50 + .5

H. D. = 20.86. H. P. = 22.68. Wt. 4.24 lbs.

4.59	77.2	75.55	23.8	105.8	
5.59	76.15	76.1	24.0	104.8	1,075

1.55 — .9

H. D. = 22.94. H. P. = 19.78. Wt. 4.24 lbs.

6.3	76.8	76.1	24.0	104.9	
7.3	81.7	76.5	24.4	104.6	1,589

.4 — .3

H. D. = 16.68. H. P. 15.63.

7.6	80.8	76.5	24.4	104.6	
8.6	78.2	76.9	24.6	104.4	1,226

.4

H. D. = 15.28. H. P. = 14.58.

8.12	77.8	76.9	24.6	104.4	
9.12	77.9	77.3	34.6	104.6	675

.4 + .2

H. D. = 15.28. H. P. = 15.97.

EXP. 225.—Rabbit; fasted 12 hours; wt. 3.68 lbs.

A. M.	A. T.	C. T.	E. T.	R. T.	LITRES.
7.15	77.3	71.1	20.4	103.2	
8.15	73.7	71.4	20.8	103.4	1,300

.3 + .2

H. D. = 12.51. H. P. = 13.12. Wt. 3.68 lbs.

8.21	73.7	71.4	20.8	103.4	
9.21	73.1	71.7	21.2	103.2	715

.2 — .2

H. D. = 12.51. H. P. = 11.96. Wt. 3.68 lbs.

8.45	70.2	67.8	19.6	102.5	
9.45	70.7	68.6	20.2	102.7	610
		<u> </u>		<u> </u>	
		.8		.2	

H. D. = 33.37. H. P. = 34.13. Wt. 4.2 lbs.

9.53	70.7	68.6	20.2	102.7	
10.53	70.	69.2	20.4	102.8	1,150
		<u> </u>		<u> </u>	
		.6		+ .1	

H. D. = 25.03. H. P. = 25.37. Wt. 4 lbs.

10.59	71.0	69.2	20.4	102.8	
11.5	71.7	69.7	20.6	101.7	715
		<u> </u>		<u> </u>	
		.5		— .1	

H. D. = 20.76. H. P. = 20.53. Wt. 3.98 lbs.

12.6	71.7	69.7	20.6	101.7	
1.6	75.5	70.15	21.9	102.5	455
		<u> </u>		<u> </u>	
		.45		+ .8	

1.15. Puncture into anterior inner end of caudate nucleus.

2.0	75.3	70.1	22.1	102.3	
3.0	75.7	70.65	21.1	102.8	390
		<u> </u>		<u> </u>	
				+ .5	

H. D. = 22.94. H. P. = 24.56. Wt. 3.94 lbs.

3.5	75.7	76.65	21.1	162.8	
4.5	73.1	71.2	21.2	103.1	390
		<u> </u>		<u> </u>	
		.55		— .7	

H. D. = 22.94. H. P. = 20.66. Wt. 3.94 lbs.

4.12	73.1	71.2	21.2	103.1	
7.12	73.0	71.6	21.6	103.5	765
		<u> </u>		<u> </u>	
		.4		+ .4	

H. D. = 16.68. H. P. = 17.98. Wt. 3.94 lbs.

5.20	73.0	71.6	21.6	103.5	
6.20	78.0	72.05	21.2	103.6	495
		<u> </u>		<u> </u>	
		.45		+ .1	

H. D. = 18.77. H. P. = 18.09. Wt. 3.94 lbs.

9.24	74.5	72.05	22.1	103.6	
7.24	74.4	72.5	22.4	103.5	475
		<hr/>		<hr/>	
		.45		— .1	
H. D. = 18.77. H. P. = 17.45.					

EXP. 227.—Rabbit; fasted 12 hours; wt. 3.84 lbs.

A. M.	A. T.	C. T.	E. T.	R. T.	LITRES.
7.50	70.4	70.4	20.9	101.2	
8.50	72.65	71.0	21.1	101.7	755
		<hr/>		<hr/>	
		.6		+ .5	

H. D. = 25.03. H. P. = 23.44. Wt. 3.84 lbs.

8.55	72.65	71.0	21.1	100.7	
9.55	73.3	71.35	21.2	100.8	1,000
		<hr/>		<hr/>	
		.35		+ .1	

H. D. = 14.60. H. P. = 14.91.

9.59	73.3	71.35	21.2	100.8	
10.59	73.4	71.8	21.3	101.0	840
		<hr/>		<hr/>	
		.45		.2	

H. D. = 18.77. H. P. = 18.40. Wt. 3.84 lbs.

11.4	63.4	71.8	21.3	101.0	
12.0	74.0	72.3	22.0	101.1	730
		<hr/>		<hr/>	
		.5		+ .1	

H. D. = 20.86. H. P. = 21.17.

12.9. 10 grains of antipyrin subcutaneously.

Wt. 3.84 lbs.

12.10	24.0	72.32	22.0	101.1	
1.10	74.2	72.75	22.1	100.6	535
		<hr/>		<hr/>	
		+ .43		— .5	

H. D. = 17.93. H. P. = 16.34. Wt. 3.84 lbs.

1.15	74.2	72.75	22.1	100.6	
2.15	74.4	73.2	22.5	100.4	685
		<hr/>		<hr/>	
		.45		— .2	

H. D. = 18.77. H. P. = 19.08.

EXP. 229.—Rabbit; fasted 12 hours; wt. 5.8 lbs.

A. M.	A. T.	C. T.	E. T.	R. T.	LITRES.
6.50	66.9	66.9	18.8	102.01	
7.50	67.8	67.8	19.6	101.9	495
		<hr/>		<hr/>	
		.9		— .1	

H. D. = 29.20. H. P. = 33.44 Wt. 5.4 lbs.

9.8	69.1	68.5	20.0	102.8	
10.8	69.2	69.1	20.2	103.0	500
		<hr/>		<hr/>	
		.6		+ .2	

H. D. = 25.03. H. P. = 26.42. Wt. 5.2 lbs.

10.15	69.2	69.1	20.2	103.0	
11.15	69.9	69.75	20.6	103.1	425
		<hr/>		<hr/>	
		+ .65		+ .1	

H. D. = 27.11. H. P. = 27.59.

11.20. 5 grains of antipyrin subcutaneously.

Wt. 5 lbs.

11.25	69.9	69.75	20.6	103.1	
12.25	70.4	702.5	20.9	103.1	459
		<hr/>		<hr/>	
		.60		.0	

H. D. = 20.86. H. P. = 20.86. Wt. 5 lbs.

12.31	70.4	70.25	20.9	103.1	
1.31	72.5	70.4	21.4	103.0	421
		<hr/>		<hr/>	
		.15		— .1	

H. D. = 6.25. H. P. = 5.77. Wt. 5 lbs.

1.35	82.5	70.4	21.4	103.	
2.35	71.8	71.7	21.8	103.3	780
		<hr/>		<hr/>	
		.3		+ .3	

H. D. = 12.51. H. P. = 13.75. Wt. 5 lbs.

2.40	71.8	71.7	21.8	103.3	
3.40	72.8	72.25	22.0	104.3	1,153
		<hr/>		<hr/>	
		.55			

Wt. 5 lbs.

3.46	72.9	72.25	22.0	104.3	
4.46	73.2	72.75	22.2	105.0	1,800
		<hr/>		<hr/>	
		.50		+ 7.	

H. D. = 20.86. H. P. = 23.76. Wt. 5 lbs.

4.53	73.2	72.75	22.2	105	
5.53	73.35	83.35	22.3	105	1,915
		<hr/>		<hr/>	
		.60		.0	

H. D. = 25.03. H. P. = 25.03. Wt. 4.54 lbs.

5.58	73.35	73.35	22.3	105.	
6.58	74.	74.00	23.0	105.1	996
		<hr/>		<hr/>	
		+ .65		+ .1	

H. D. = 27.11. H. P. = 27.59.

EXP. 230.—Rabbit; fasted $3\frac{1}{2}$ days; same animal as in Exp. 229; wt. 4.78 lbs.

A. M.	A. T.	C. T.	E. T.	R. T.	LITRES.
8.15	68	67.9	18.3	102.3	
9.15	74	68.6	19.3	102.0	1,273
		<hr/>		<hr/>	
		+ .7		— .3	

H. D. = 29.20. H. P. = 28.26.

9.19	74	68.6	19.3	102.0	
10.19	72	69.2	19.4	101.8	1,227
		<hr/>		<hr/>	
		.6		— .2	

H. D. = 25.03. H. P. = 24.39. Wt. 4.76 lbs.

10.35	72	69.2	19.4	101.8	
11.25	74	69.6	20.0	102.1	1,360
		<hr/>		<hr/>	
		.4		+ .7	

H. D. = 16.68. H. P. = 18.64.

11.31	74	69.6	20.	102.1	
12.31	72	70.25	20.5	102.1	1,275
		<hr/>		<hr/>	
		.65		.0	

H. D. = 27.11. H. P. = 27.11.

12.32. Rabbit received 5 grains of antipyrin subcutaneously.

12.35	72	70.25	20.5	102.1	
1.35	73	70.50	20.9	102.2	110
		<hr/>		<hr/>	
		.25		+ .2	

H. D. = 10.43. H. P. = 11.23. Wt. 4.74 lbs.

1.40	73	70.5	20.9	102.3	
2.40	75	71.0	21.1	102.8	1,100
		<hr/>		<hr/>	
		.5		+ .5	

H. D. = 20.86. H. P. = 22.82

2.46	75	71.0	21.1	202.8	
3.46	72	71.6	21.3	103.5	741
		<hr/>		<hr/>	
		.6		+ .7	

H. D. = 25.03. H. P. = 27.78

3.51	72	71.6	21.3	103.5	
4.51	72	72.5	22.0	103.4	354
		<hr/>		<hr/>	
		.9		— .1	

H. D. = 37.54. H. P. = 37.16.

EXP. 236.--Rabbit; fasted 3 days; wt. 3.30 lbs.

A. M.	A. T.	C. T.	E. T.	R. T.	LITRES.
8.12	74	72.3	72.52	102.6	
9.12	74	72.7	72.88	102.5	897
		<hr/>		<hr/>	
		.4		.1	

H. D. = 19.32. H. P. = 19.04. Wt. 3.30 lbs.

9.21	74	72.7	72.98	102.5	
10.21	74	73.2	73.50	102.3	1,030
		<hr/>		<hr/>	
		.5		.2	

H. D. = 24.15. H. P. = 23.60. Wt. 3.30 lbs.

10.30	74	73.2	73.50	102.3	
11.30	74	73.7	73.78	101.6	1,055
		<hr/>		<hr/>	
		.5		— .7	

H. D. = 24.15. H. P. = 22.12. Wt. 3.30 lbs.

11.40	74.	74.7	73.28	101.6	
12.40	73.	74.	74.28	101.7	270
		<hr/>		<hr/>	
		.3		+ .1	
	H. D. = 14.49.		H. P. = 14.76.		

12.45. 10 grains of antipyrin.

Wt. 3.30 lbs.

2.48	73.	74.	74.28	101.7	
3.48	78.5	74.6	74.16	100.8	2,040
		<hr/>		<hr/>	
		.6		— .9	
	H. D. = 28.98;		H. P. = 26.62. Wt. 3.38 lbs.		

1.56	78.5	74.6	73.24	100.8	
2.56	75.	74.95	75.4	101.1	1,040
		<hr/>		<hr/>	
		.35		+ .3	
	H. D. = 16.90.		H. P. = 18.53. Wt. 3.28 lbs.		

3.3	75.5	74.95	74.56	101.1	
4.3	76.0	75.4	76.12	101.3	1,065
		<hr/>		<hr/>	
		+ .45		+ .2	
	H. D. = 21.73.		H. P. = 22.54. Wt. 3.28 lbs.		

4.10	76.	75.4	76.12	101.3	
5.10	75.5	75.8	76.48	102.6	1,111
		<hr/>		<hr/>	
		.3			
	H. D. = 19.42.		H. P. = 20.13.		

EXP. 237.—Rabbit; fasted 3 days; wt. 2.90 lbs.

A. M.	A. T.	C. T.	E. T.	R. T.	LITRES.
8.24	71.	72.1	72.34	102.0	
9.24	72.	71.3	72.88	101.7	809
		<hr/>		<hr/>	
		.2		— .3	
	H. D. = 9.6.		H. P. = 8.94. Wt. 2.80 lbs.		
9.30	72.	72.2	73.06	101.7	
10.30	72.	72.6	73.42	101.2	948
		<hr/>		<hr/>	
		.3		.5	
	H. D. = 14.49.		H. P. = 13.33. Wt. 2.78 lbs.		

10.37	72	72.6	73.42	101.2	
11.37	73	72.9	73.50	101.	830
		<u> </u>		<u> </u>	
		+ .3		— .2	

H. D. = 14.49. H. P. = 14.03. Wt. 2.78 lbs.

11.48	72	72.9	72.50	101.	
12.58	76	73.25	74.28	100.9	
		<u> </u>		<u> </u>	
		.35		— .1	

H. D. = 16.90. H. P. = 16.67.

1.5. 10 grains of antipyrin subcutaneously.

Wt. 2.74 lbs.

1.11	76.5	73.2	74.16	101.2	
2.11	74.0	73.5	74.28	100.9	
		<u> </u>		<u> </u>	
		3.3		.3	

Convulsive effect of antipyrin present.

H. D. = 14.49. H. P. = 13.81. Wt. 2.72 lbs.

2.18	74	73.5	74.25	100.9	
3.18	76	73.75	74.28	99.6	1,245
		<u> </u>		<u> </u>	
		.25		— .3	

H. D. = 12.07. H. P. = 11.40. Wt. 2.72 lbs.

3.26	76	73.75	74.28	99.6	
4.25	76	73.95	64.68	99.9	1,165
		<u> </u>		<u> </u>	
		.20		+ .3	

H. D. = 9.66. H. P. = 10.33. Wt. 2.72 lbs.

4.33	76	73.95	74.68	99.9	
5.33	76	64.225	74.86	100.2	1,026
		<u> </u>		<u> </u>	
		.275		+ .3	

H. D. = 13.28. H. P. = 13.95.

Reviews.

LECTURES ON NERVOUS DISEASES. By Ambrose L. Ranney, A.M., M.D. P. 775. F. A. Davis, Philadelphia, 1888.

Dr. Ranney, in this work, heralds a new departure in medical literature by profusely illustrating his text in colors. This will unquestionably catch the eye of the general practitioner, and probably this was the publisher's main object in view, but nevertheless the colored diagrams in many instances have a decided advantage over uncolored ones. It is true that ordinarily it makes little or no difference, except from an æsthetic standpoint, whether an illustration is colored or not; but in diagrams representing fibre system tracts, or where it is necessary to differentiate between motor and sensory fibres, or where association system of fibres cross, the distinguishing of these different tracts by prominent colors materially aids the facility of comprehension. The work is divided into seven sections, two of which deserve more than a passing notice. These are sections V. on "Functional Nervous Diseases," and section VII. on "Electricity in Medicine, and a Glossary of Neurological Terms." The other sections, on Diseases of the Brain, Diseases of the Spinal Cord, Toxic and Unclassified Nervous Diseases, the Examination of Patients, and the Anatomical, Physiological and Pathological Deductions Respecting the Nerve Centres, are written with the author's characteristic clearness and attention to details. Doubtless many neurologists will take exception to the few defects which exist, and will condemn the work on that account. If the volume was intended as a text-book for neurologist a closer scrutiny would be pardonable, but the intention of the author was evidently to reach the greater mass of physicians in general practice who would care little or nothing for the anatomical or physiological errors so long as they were taught the nature, diagnosis, and treatment of the various nervous diseases they are called upon to treat. Errors unquestionably exist in this book, as they do in most other books. Let us take, for example, the diagram of the optic thalamus (Luys') on page 23. Luys' claim for four special centres in the thalamus cannot be substantiated by the most careful observer. The diagram is manifestly incorrect, and should never have been reproduced. To the neurologist this and similar defects are probably unpardonable, but the average general practitioner, we regret to say, does not know or care anything about the minute structure of the thalamus. As long as a faithful description of disease is given, with the proper therapeutical measures for its relief he is satisfied, and from this standpoint Dr. Ranney's work must necessarily be eminently successful.

Section V. deserves special notice for the vigor of the author's championship of Dr. Steven's views relative to anomalies of the visual apparatus as a cause of functional neuroses. Under the heading of "Functional Neuroses," the author includes a certain

percentage of epilepsy, chorea, hysteria, and hystero-epilepsy, neurasthenia, migraine, neuralgia, and in some cases, imperfect performance of some of the functions of the abdominal and thoracic viscera. For a number of years there has not been any question in the minds of most neurologists in regard to the influence of reflex irritation as a factor in the production of the diseases classified by Ranney as "functional neuroses;" but it was not until Dr. Stevens's paper was published that neurologists were lead to investigate anomalies of the visual apparatus to any great extent. Dr. Ranney was one of these investigators, and his researches have led him to a full concurrence in Dr. Stevens's views. In this respect he probably stands alone among neurologists, as he himself admits, but his observations and deductions cannot fail to encourage more careful and thorough examinations of the eye in cases of nervous diseases; and though the evidence, considered from both sides, does not seem to warrant us in accepting Dr. Ranney's views in their entirety, yet, unquestionably, in well selected cases, they are incontestable.

In the seventh section the subject of Electricity in Medicine is treated with more thoroughness than in any other work on nervous diseases. The description of the numerous and necessary pieces of apparatus is complete, and leaves nothing to be desired. Electrical dosage, the manner of application, and the affections for which electricity is serviceable, are all considered carefully and scientifically, and show unusual familiarity with a subject, the knowledge of which is becoming more and more of daily importance.

The glossary of neurological terms is also an innovation which will be of material assistance to many readers. As a whole, Dr. Ranney's work will commend itself to the general profession, and will be found on many tables where perhaps more celebrated works are unknown.

**Which is the Most Powerful ?
and the Most Reliable ?
of All Pepsins ■**

Merck's Pepsin 1:2000 !

— SCALE OR POWDER —

SEE "MERCK'S INDEX," PAGES 106 AND 167

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

ABSCESS OF THE BRAIN—OPERATION—DEATH
ON THE NINTH DAY.

By J. T. ESKRIDGE, M. D.,

DENVER, COLO.

THE practical side of cerebral localization has attained to such importance as to demand that every case illustrating points in the diagnosis and treatment of lesions of the brain amenable to surgical interference should be published in full, no matter what the mistakes have been or how the case has terminated.

J. P., male, æt. 30, Colorado ranchman, contracted typhoid fever in the latter part of September 1887. By November 1, he was fairly convalescent from the fever, but the disease had left him with a purulent discharge from the right middle ear. Early in November his physician ceased to visit him, and he had no further medical attention until the evening of November 20th, when he was found in bed and delirious. It was learned that from early in November, when last seen by his physician, to about the middle of the month, he had appeared to be doing well, eating heartily and gaining in strength. About November 15, his appetite became indifferent, and he complained much of headache. He was observed to be feverish and irritable. Symptoms of ill-health gradually became more pronounced, and he had been slightly delirious for two or three evenings before his physician was called to see him.

November 20, 8 P. M., T. 101°; P. 100; R. 24. He was delirious and delusional, seeming to think that his attendants were trying to kill him. The muscles that move the left wrist were paralyzed. I was requested to see him the next day (Nov. 21) at 11 A. M., in consultation with his attending physician. We found him lying quietly in bed and intently observing the movements of every one in his room. His eyes followed us when we walked from one side of the room to the other, as one suspicious of the actions of those by whom he is surrounded. He said nothing unless some one approached his bed, when a shower of profane abuse followed. He recognized his attending physician and seemed to appreciate his efforts to relieve him, but was disturbed and irritated by the presence of any one else. He accused his attendants and me of seeking an opportunity to kill him, and damned us accordingly in the unpolished language of a western ranchman. The muscles of the left angle of the mouth were paretic. This angle of the mouth was neither depressed nor elevated when he was quiet, the usual fold was obliterated, but the depressor and elevator muscles seemed to contract slightly when he was talking excitedly. There was no aphasia. The extensor muscles of the left hand and wrist were paralyzed and flaccid, and flexors were paretic. When the arm was held up and the hand balanced at the joint it would invariably, without any apparent resistance, fall forward to the prone side of the forearm, but if the arm was carried a little further backward, and the weight of the hand thrown on the flexor muscles of the wrist, the hand slowly assumed an extended posture, and his efforts to again flex the hand were unsuccessful so long as the arm was held in this position. When the arm was lying at rest the fingers and hand were flexed. The other muscles of the left arm and the left leg muscles were unaffected. The tongue in protrusion deviated slightly to the left. There was an abscess in the cellular tissue of the right forearm, but there was no impairment of muscular movement on the right side. His mental condition precluded the possibility of accurately testing the special senses, or of getting from him an account of his

own feelings. I received profane abuse for every question that I put to him. On roughly testing vision it was found that he could see with either eye. Pinching or pricking over the paralyzed and paretic muscles was readily recognized. It was not possible to determine whether the sense of touch in these parts was present. There was a sanious or semi-purulent discharge from the right ear, slight in amount but offensive in character. The tissues over the right mastoid process were neither reddened, swollen, nor tender to pressure. Temperature 101° ; pulse 110° ; respiration 24. He could not be induced to hold his head still, so that I was unable to get the head temperatures. The tongue was coated and bowels constipated, and he would take nothing but a little milk. He had not slept much during the last two or three nights, and presented a depressed and anxious appearance.

I diagnosed abscess in the upper portion of the lower third of the post-central convolution (ascending parietal), stated that I thought the corresponding portion of the pre-central convolution and the centre for movements of the tongue were being involved, either from pressure or extending inflammation, and advised trephining at the earliest opportunity. For several reasons the operation was not performed until two days afterwards. I saw him the next day, but there was no apparent change in his symptoms. Just before the operation I again examined him. The left wrist and hand muscles appeared about the same as when I first saw him, two days before, but the muscles of the left angle of the mouth showed less weakness, and the tongue deviated but little, if at all, from the median line. T. 101.5° ; P., 110; R. 30.

Before giving an account of the operation and the subsequent progress of the case, it is important to state that the patient's surroundings were the most wretched and unfavorable imaginable for a delicate surgical operation. He was with acquaintances who kept a ranch boarding and lodging house just outside of town. The food was coarse, poor, and badly prepared. He had no regular attendant, and the attention he received was ignorant, irregular and

uncertain. The room was small, filthy and occupied by one or two other lodgers. It contained a small wood stove, and sometimes the temperature of the room was raised to 80° or 90°, and at other times allowed to cool down to 35° or 40°, the outside temperature at the time being considerably below zero. Under such circumstances it was impossible to secure ventilation. At noon of November 23d, the patient was etherized, the scalp shaved and disinfected with a solution of bichloride of mercury (1 to 2000). On account of the resistance offered by the patient, it was necessary to etherize before undertaking to shave the scalp. The surgeon was careful to cleanse and disinfect instruments, sponges, and the hands of all the assistants. I indicated the seat of the abscess, using the lines given by Seguin in "Pepper's System of Medicine," (vol. v., p. 93). The largest size trephine was used and a disc of bone was skillfully removed without injuring the the dura mater. The membrane was not abnormally adherent, and did not seem inflamed. It protruded into the opening as soon as the bone was removed. The protruding part gave a soft, semifluid sensation to the touch. After all oozing from the scalp wound had been stopped, the dura was opened, when a small quantity of serous fluid, but no pus, escaped. The outer cortical brain substance and the pia appeared nearly normal in color, but on palpation the sensation was like that given by a semifluid mass. On cutting into the brain substance, about one-eighth to one-quarter of an inch in depth, nearly an ounce of pus and detritus of broken down brain substance was evacuated. The abscess cavity was wiped, but not washed out, and was partially filled by means of a small piece of disinfected sponge, a drainage tube was inserted, a few stitches of fine silk were placed in the scalp, leaving a place for drainage through which a finger could be passed, the wound was covered with lint soaked in a solution of bichloride of mercury, and over this a bandage was placed.

I was informed by the attending physician that on the evening of the day of the operation, after the effects of the anæsthetic had passed off, that the patient was bright and

less irritable, free from delirium and did not seem to have much increase of temperature above the normal. I saw him at 11 a. m., the day after the operation and noted marked improvement. The flexor muscles of the left wrist were stronger than before the operation, the extensors were still completely paralyzed, the tongue was protruded in the median line, the left angle of the mouth showed no preceptible difference from its fellow, the irritability of temper was greatly diminished, he showed no aversion to attendants or strangers, talked freely about his condition and answered questions intelligibly. He had slept well the night following the operation and said that he felt much better than he did before it was performed. The right ear had been kept cleansed. As there was no apparent discharge from the wound and no odor, the surgeon decided not to disturb the dressing that day. The patient's temperature had fallen from 101.5° to 99°; pulse from 110 to 90; respirations from 30 to 24. At this stage of the case the weather became intensely cold and stormy, and as my lung trouble was more or less active, I did not dare to venture out to see him again until the fifth day after the operation. I learned that on the second day after the operation the patient seemed bright and cheerful, he had no delirium or delusions, ate and slept well, and his temperature and pulse was nearly normal. The wound was dressed, but the abscess cavity was not washed out. There was but little discharge, and this was not offensive. The discharge from the ear was odorless and had nearly ceased. After this the surgeon who had operated did not have an opportunity to see the patient again for two or three days. He was left in charge of the attending physician, who changed the dressings daily, but did not wash out the abscess cavity. On November 27th, five days after the operation, we were requested to see the patient again, as his fever had returned and he was again delirious. We found his temperature 101°; pulse, 120; respiration, 32. He was semi-unconscious and seemed to be in a stupor. The discharge from the brain had increased and become very offensive. The abscess cavity was thoroughly washed out and kept as clean as possible, but complete cleanliness,

on account of the ignorance of the attendants and the distance at which the man lived from his physician, was not kept up. Fever increased to 102° , the patient gradually became deeply comatosed and died December 1st, nine days after the operation. I was not able to see him from the fifth day after the operation until after his death. I learned that the left side had become completely paralyzed and limp, and on the day before death the muscles of the right side of the chest and of the right leg kept up a more or less constant twitching. It was with difficulty that we obtained permission to make an autopsy, and this was not granted until the second day after death, while the friends were assembled to attend the funeral services.

The brain only was allowed to be examined. The odor of the discharge from the brain and ear was horribly sickening. The adhesions of the dura to the skull cap were slightly increased over nearly the entire convex surface and external aspect of the right hemisphere, and over the left hemisphere along its median or longitudinal surface. On removal of the dura, the inner side of which was covered with pus, the arachnoid, pia and brain substance of the right side seemed to be one mass of pus from the middle of the frontal lobe to the parieto-occipital fissure. Over this area the brain substance was softened to the depth of about one-quarter of an inch. A cavity, the seat of the abscess, was found corresponding to the opening in the skull, but owing to the great amount of softening it was impossible to positively determine what convolution it occupied. On comparing it with the post-central convolution on the left side it corresponded to about the junction of its middle and lower third. Over the convex surface of the left hemisphere the membranes and brain substance seemed nearly normal in appearance, except around the upper end of the fissure of Rolando, where the membranes were injected and inflamed. The affected portion of the membranes extended longitudinally from about one inch posterior to the upper end of the fissure of Rolando to an inch in front of it, and laterally from the longitudinal sinus to a distance of half or three-quarters of an inch. The brain substance

beneath this area was slightly softened. On separating the hemispheres at the longitudinal fissure, about half a teaspoonful of creamy pus was found, and the membranes on the median surfaces of the hemispheres were inflamed and the brain substance softened, but to a greater extent on the right than on the left side. The greatest amount of softening and inflammation of the median surface of the left hemisphere was over the paracentral lobule. At the base of the brain a small quantity of pus was found on the petrous portion of the right temporal bone, around which existed a small area of meningeal inflammation. The remainder of the base of the right hemisphere and the whole of the base of the left hemisphere presented a nearly normal appearance. No time was allowed to examine the condition of the internal portion of the right petrous bone or of the mastoid cells. Much of the softening of brain substance which had taken place was undoubtedly a post-mortem result.

In reviewing this case in the light of the present thorough antiseptic method of treating surgical abscesses of the brain, it seems to me that a brilliant result was sacrificed to ineffective after-treatment. Much of this was unavoidable, because of the poverty of the patient, his miserable surroundings, and the difficulty in getting proper medical care, owing, to some extent, to the inconvenience his physician was put to in visiting him.

Twitching of the muscles of the right leg and the right side of the chest the day before death and finding at the autopsy meningitis, on the left side, largely limited to the paracentral lobule and the extreme upper portions of the post and pre-central gyri are phenomena in harmony with the results of the experiments in cerebral localization made by Horsley, Schäfer and others.

Drs. Horsley and Beevor place the spot where they believe extension of the wrist is most represented in the anterior border of the upper limb region. The case I have reported would seem to point to the post central convolution as the seat of this function in the present instance.

CONTRIBUTION TO THE STUDY OF ANÆSTHETIC LEPROSY, WITH SPECIAL REFERENCE TO PARTIAL SENSORY DISORDERS.¹

By DR. GEO. W. JACOBY.

IT is very far from my purpose to even refer, before this society to the work and investigations which have been so energetically devoted during the last few years to the study of leprosy in its general aspect. The literature of this subject is so large, and the names of investigators in this field so renowned, that it would seem almost superfluous to endeavor to add anything to our present knowledge. But these remarks are true only from a dermatological standpoint; if we examine the question from its neuropathological aspect we still find questions which have been insufficiently ventilated, and upon which, notwithstanding the recent attention of Schultze and others, our knowledge could easily be enlarged. The points which are still *sub judice*, and which should be carefully noted in every case of so called anæsthetic leprosy are: the electrical excitability of the affected muscles; the condition of the reflexes; the absence or presence of fibrillary twitchings and, above all a thorough examination of the sensory symptoms should be made.

These data are important for the purpose of deciding two main questions; firstly, whether in a given case the leprosy changes affect the central nervous systems, and secondly, whether we are actually dealing with a case of leprosy; for if in a case of supposed anæsthetic leprosy, in addition to the usual symptoms, progressive atrophy of muscles and trophic disturbances, such as ulceration and gangrene, we also find partial sensory disorders instead of the usual anæsthesia, we can easily understand how the differential diag-

¹ Read before the American Neurological Association, Long Branch, N. J., June 27, 1889.

nosis between leprosy and syringomyelia may cause considerable difficulty. As a proof of the fact that these assumptions are not purely gratuitous ones, but that such a combination of symptoms as here mentioned does occur, I will describe the following case, the opportunity of examining and reporting which I owe to the kindness and courtesy of Dr. F. C. Valentine of New York.

C. S., male, æt. 18. Birthplace, Cuba. When eight months of age he was removed to Cartagena, U. S. of Colombia. His parents are alive and in good health. They have had eight children, five boys and three girls. The girls are all dead. One of the boys, during several years had some skin disease, but is now well. No history of heredity in collateral branches. Grandparents were over seventy when they died.

The patient himself was always delicate. He was not nursed by his mother, but by a Cuban woman, who subsequently had an illegitimate child, which at the age of six years developed a similar disease to that with which our patient is now afflicted. From his third to seventh year the patient was perfectly healthy. At this time an eruption appeared upon the buttocks, consisting simply in a reddening of the skin, which being observed by the mother, she asked the boy whether he had been struck. This was not preceded by pains. Similar spots hereupon appeared on the thighs, arms and forearms, lasted about a year and then disappeared. All of these spots looked as though he had been struck a slight blow sufficient to redden the skin. They did not have any definite form and varied in size, some being very small, others half an inch in diameter. Just prior to the appearance of the first spot, patient found a place upon one of his legs, near the tibia, into which he was able to insert pins, without producing pains. He did this purely "for fun," because some of his playmates could also do it. When eight years of age the spots disappeared, and remained absent for a year. They then again became apparent, and extended to the face, involving the chin, face and ears; they again disappeared without leaving any mark. During this time he was under treatment. These

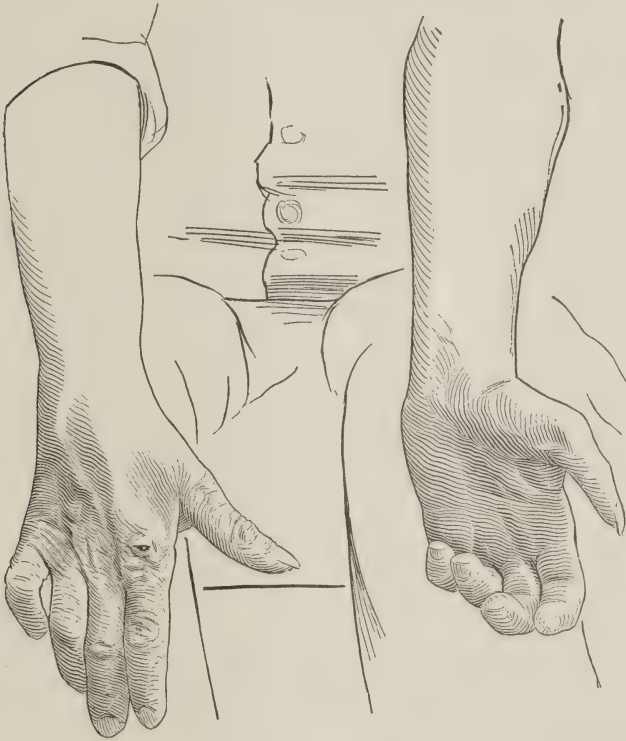
spots, when the patient was ten years old again reappeared, began to swell and became elevated and glossy. Coincidentally his ears assumed a dark brownish red appearance, and became swollen and deformed. The hairs on the spots upon the body, fell out. The spots showed no distinct line of demarcation from the surrounding territories. When eleven years old, he suffered from restlessness at night, not



being able to find a comfortable position for his limbs ; also suffered from frequent cramps in the calf muscles. At the age of twelve, "a corn" as he calls it, appeared at the ball of the great toe on the right foot, ulcerated and made a deep sore. The same then occurred on the left foot. From this time until his fifteenth year, he was occasionally better, occasionally not so well. At fifteen, his hands became swollen, also the skin of the thorax, the lobes of the ears

and the nasal mucous membrane. The hands were not discolored, but the chest, chin and ears were brownish red. The joints of the fingers were enlarged but painless.

On the fourth finger of the right hand at the second joint, a swelling appeared which suppurated and left an ulcer. During all this time the ulcers on the feet persisted. At the age of sixteen he was attacked by some form of fever



and remained in bed for a month. During this time, all symptoms of disease again disappeared, only to reappear soon after getting out of bed. It was at this time that he noticed that he could not grasp small objects, could not button his clothes, and frequently burned his hands without being aware of it. Now, also the small muscles of the hands began to waste ; wasting proceeded slowly. Fingers continued to swell and finally began to contract in a pos-

ition of flexion. Previous to this, he had a large number of "corns" upon his fingers. At various times pieces of bone came from the ulcers on his feet.

Status præsens, Aug. 8, 1888.—Complains chiefly about the ulcers on his feet, and of the appearance of his hands. He says that his hands do not incommode him much, although he has had to give up his trade of watchmaking. Medium height, weight 125 lbs. Head and face normal; eye-brows sparse; no alopecia. On the right upper part of the chest $1\frac{1}{2}$ inches below the clavicle, is a circular scar with central della and striæ radiating to the periphery. Several such scars on chest and scars of different character (probably from setons) on arms.

The hands show marked atrophy of the interossei muscles and of the eminences, most marked on dorsal surface between thumb and finger. There is contracture of the flexor tendons, most marked in the small finger of both hands, main en griffe on both sides. There is no scar at the contractures. Joints freely movable. The thickened ulnar nerves can be plainly felt through the skin in the epitrochlear regions. Pressure over the nerves produces pain, which is also felt in the fingers. The skin of the palms is dry and shining. Growth of nails retarded. Wasting of muscles of forearms, also of deltoids. The toes are malformed. The feet are plump and stumpy and show but little motion. The arch of the foot is lost. On the ball of each foot is seen a deep suppurating ulcer of about the size of a five cent piece. Also in the middle of the plantar surface of each foot, over the carpo-phalangeal joint is an ulcer in the process of healing. Motion of the thighs is good. (See photographs of hands and feet).

Electrical examination of the affected muscles shows a reduced excitability to both currents. Distinct reaction of degeneration could nowhere be obtained, but in the left hand, the muscles supplied by the ulnar nerve show ACC=KCC. The reaction of the apparently normal muscles is normal.

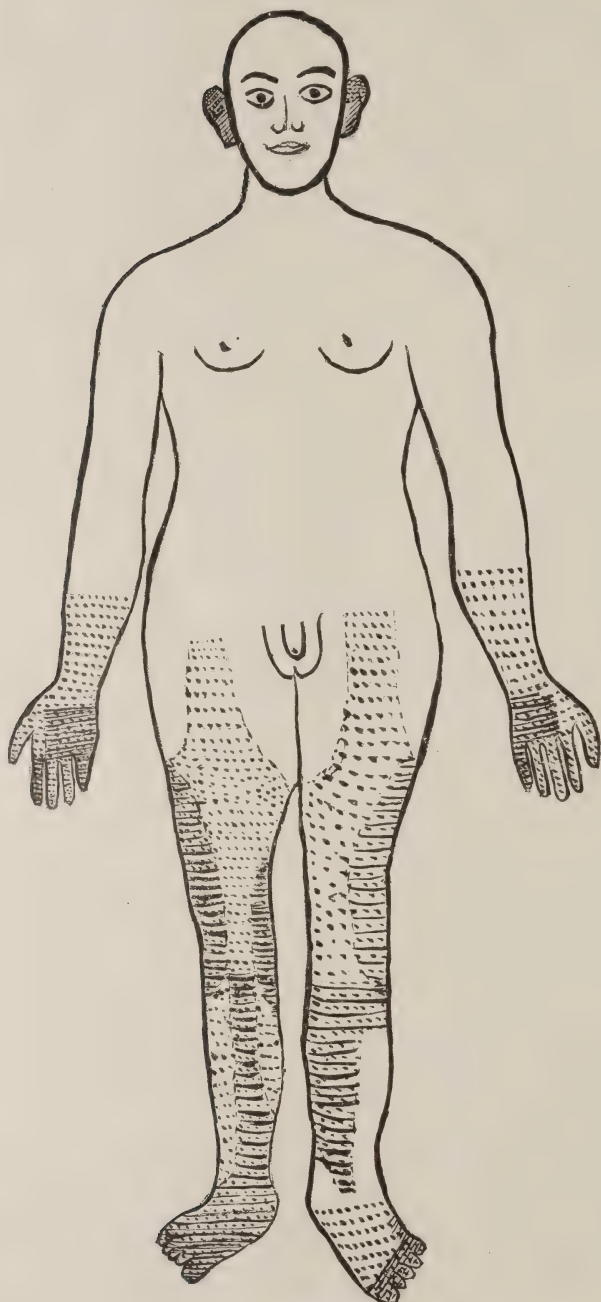
Mechanical excitability of muscles normal. *Reflexes*: the patellar tendon reflexes are much increased on both sides. Cremaster and abdominal reflexes present.

Sensation—Tactile sensibility.—The examination was made with a pin, with cotton and by pressure with the finger. Both arms, shoulders, and back showed reduced sensation (*hypæsthesie v. Rentz*) in irregular patches. In these places the perception for all strong applications was perfectly clear. Weak applications were also noticed and correctly localized, but the differentiation by the patient of the various modes of procedure was not plain; thus, if only lightly touched, the head of the pin could not be distinguished from the point, and cotton not from the finger. In the entire remainder of the body, the sense of touch is perfectly normal.

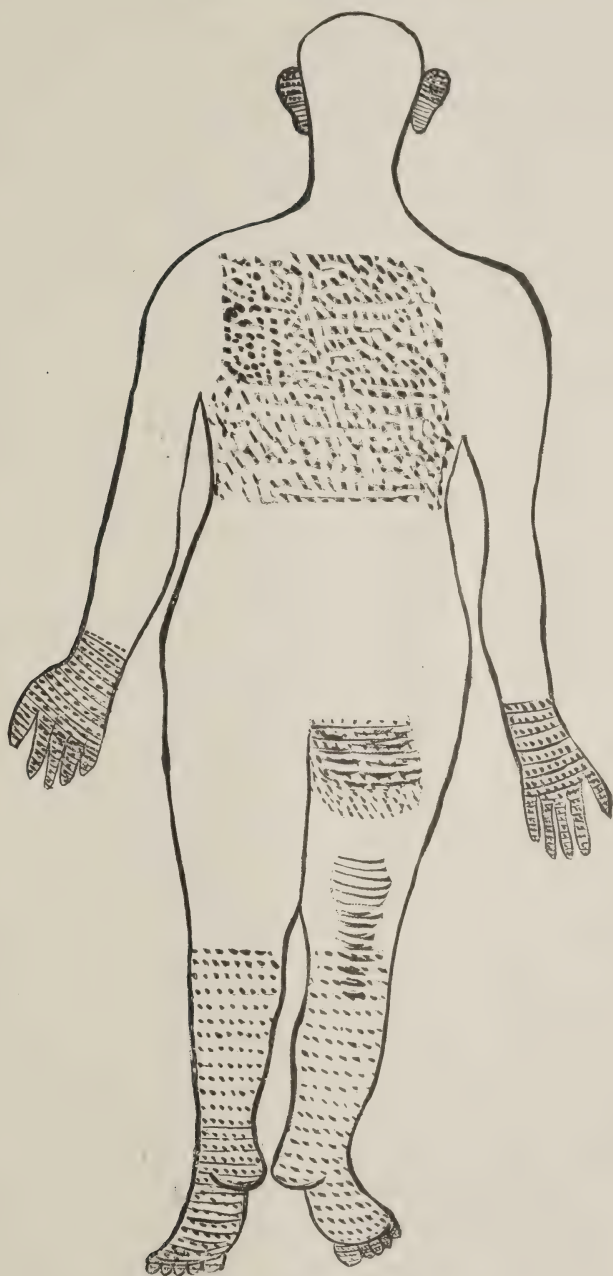
Sensibility to pain.—Large territories of pronounced analgesia are found. In many of these territories it is possible to transfix an entire fold of skin, the patient saying that he feels it distinctly but it does not hurt. The analgesic territories are: both ears, entire dorsal surface of hands; on the palmer surface, the little finger and the ulnar side of the palm upward to above the middle of the hand and inward just beyond the medium line; also the entire palmar surface of the index finger. This distribution is the same for both hands. Also analgesic is the left thigh internally and externally, from the knee upwards to near the middle. On the right thigh, in addition to the same territories being affected, an area internally at the bend of the knee was involved, and also a large area involving the entire gluteus maximus and the lower part of the territory supplied by the *nervus cutan. femoris post.* On the *left leg*, the upper half of the saphenous territory and dorsal surface of the toes, and on the *right leg* the territory supplied by the *nerv. peroneus superf.*, and the entire dorsum of the foot and toes were involved. The entire plantar surface of the left foot, and the plantar surface of the toes on the right foot, were also affected.

The first examinations of the *temperature sense* were made with hot and cold water as it came from the pipes, and it was thereby demonstrated that there was complete loss of temperature sense in the following territories:

The upper part of both ears, the dorsal surface of both



..... Disordered temperature sense.
== Analgesia.



..... Disordered temperature sense.
== Analgesia.

hands, the palmar surface of the hands, the wrists, lower half of forearms, nearly all of the crural and lumbo-inguinal territories on both sides of the anterior surface of the thighs, both dorsal surfaces of the feet, entire posterior part of the legs and the plantar surface of the feet. In addition to these parts, the entire anterior surface of the right leg and the gluteal region of the same side were affected.

It will thus be seen, by reference to the annexed chart, that the distribution of the territories showing disordered temperature sense is much more symmetrical than that of the areas of analgesia. We are also impressed by the fact that neither the analgesic areas, nor those with disordered temperature sense, are limited to the distribution territories of particular cutaneous nerves; we also must remark that although the same areas which show loss of pain sense also show loss of temperature sense, still the loss of temperature sense also affects territories which in other respects are normal. Subsequently, further examinations of the temperature sense were made with much lower temperature, and it was then seen that in the ulnar and median territories of both hands, the sense for very low temperatures was preserved. These examinations were made with test tubes filled with ice, with cooled water and with hot water.

The result of these examinations was that water at 110° F. was described as warm, while blisters could be produced without eliciting any complaint of heat. Water between 50° and 110°, was described as indifferent, neither warm nor cold, but temperatures below 50° were described as cool and ice as cold.² Sense of posture and position of limbs normal.

Trophic disorders.—In addition to the ulcers already described, the patient asserts that he never sweats. This could not be verified, for a subcutaneous injection of 0.02 pilocarpini muriatici, produced perspiration of the entire body. The hairs on the body are sparse; here and there very long silk-like hairs are found. Thus, on the middle

² Normally from 86 upward is considered warm and 75 and below, cold.

anterior surface of the right arm is one of about 8 inches in length.

No fibrillary twitchings, no spontaneous pains. Eyes, sense of taste, speech, smell, and hearing, normal.

Internal organs normal.

If now we briefly summarize the case before us we must lay stress upon the following features :

1. Marked atrophy of both hands, principally of the small muscles, but also affecting the forearms and deltoids.
2. Increased tendon reflexes.
3. Sensory paresis, consisting chiefly of analgesia and disordered temperature sense, with preservation of tactile sense.
4. Trophic disorders.

If in addition to these points we consider the previous history of the case, together with the etiological data, we can, I think, arrive at no other conclusion than that we are dealing with a case of leprosy, of the so-called nerve or anæsthetic form.

The symptoms presented by this case do not essentially vary from those described by other writers ; certain changes to which little attention has heretofore been paid, have been carefully examined, and these changes, judging from the few cases in which they are referred to at all, do not appear to be of constant occurrence in this disease. Thus, Breuer, Rosenthal,³ Vallin and Rosenbach⁴ also found the tendon reflexes increased, while Schultze⁵ did not find any abnormal deviation. Leloir⁶ mentions the presence of fibrillary twitchings in one of his cases, while this symptom was absent in mine, and as far as I know in all other reported cases. Reaction of degeneration was not found by Rosenthal, Rosenbach, or myself, but was present in cases of

³ M. Rosenthal. Zur Klinischen Charakteristik der Lepra Anaesthetica. Vierteljahrschrift fuer Dermatologie, p. 425, 1881.

⁴ Rosenbach. Ueber die Neuropathischen Symptome der Lepra. Neurol Centralblatt., p. 361, 1884.

⁵ Schultze, F. Zur Kenntniss der Lepra. Deutsches Arch. f. Klin. Med.izin., vol. 43, 1888, p. 496.

⁶ Leloir. Traité de la Lepre.

Dehn⁷ and Schultze. Loss of temperature sense was observed in the cases of Rosenthal and Rosenbach, but could not be found by F. Müller⁸ nor Schultze. The symptoms here mentioned are of particular value in deciding upon the probable anatomical location of the disease, and the fact that these symptoms vary in the different cases would of itself lead us to suppose that the location of the pathological process is not always the same, but that this also varies according to circumstances. In one case, therefore, it is certainly admissible, if we consider the symmetrical distribution of the atrophy and of the trophic disorders, the increased tendon reflexes, and the partial loss of sensation, to doubt that the assumption of a peripheral neuritis constituting the entire anatomical change is warranted, notwithstanding the fact that Leloir,⁹ Schultze, and the majority of writers take this view of the matter.

Schultze explains the increased tendon reflexes, when they occur, by assuming an abnormal irritability of the muscles, and also believes that the symmetrical distribution of the disease in the hands, feet, and face is due to the greater exposure of these parts to direct infection. That partial sensory disorders have been found in anæsthetic leprosy, Schultze does not deny, but states positively that he has never found any. All such assumptions and negative statements go for nothing in the face of positive facts, and these facts all tend toward corroborating the statement already made, that the assumption of a peripheral neuritis alone is insufficient to explain the symptoms encountered.

It will also at once become evident that this question of the central or peripheral localization of the pathological process in the anæsthetic form of leprosy, must assume practical interest, on account of the relationship which this class of cases may bear to cases of syringomyelia.

⁷ Dehn. *Deutsche Medizin. Wochenschrift.*, 1887, No. 45, p 988. Discussion of a paper by Dr. Arning.

⁸ Müller, F. Ein Fall von Lepra. *Deutsches Archiv. für Klinische Med.*, vol. 34, p. 205.

⁹ Leloir. *Gazette des Hopitaux*, p. 575, 1888.

Were it not for the etiological factors and previous history of my case, we would be in a quandary as to the differential diagnosis between leprosy and syringomyelia, provided, of course, that the symptomatology of the latter affection as to-day accepted, may be considered sufficient for diagnostic purposes. Attention has also been called to the possibility of mistaking these two affections, by other writers, and in practice this difficulty is illustrated by the case of Steudener¹⁰ and Langhans.¹¹ In both of these cases the diagnosis of leprosy was made *intra vitam*, and the autopsy disclosed cavities in the cord.

In Steudener's case the following was shown on Autopsy. Spinal cord. A fissure like cavity, filled with a mucous sticky fluid, extends with various interruptions from the medulla to the lumbar enlargement. A loss of substance is found in the pyramid and a larger one near the vagus nucleus. A horse-shoe shaped, fissure like defect is also found above the cervical enlargement. Peripheral nerves of right upper extremity show thickening of the neurillemma; foci of small granulation cells and spindle-shaped swelling of the right radial nerve.

In Langhans' case, the autopsy showed an immense focus of softening, implicating the posterior horns, Clarke's columns and the grey commissure. Here is found a cavity which traverses the cord in a transverse direction. The peripheral nerves showed thickening of the peri and endoneurium.

These cases then, which from their clinical history were looked upon as cases of leprosy showed upon autopsy in addition to changes of the peripheral nerves such as we find in leprosy, changes in the spinal cord such as we find in syringomyelia.

Even assuming that these cases were not cases of leprosy at all, but were true cases of syringomyelia, as Schultze contends, there is still sufficient evidence from

¹⁰ Steudener, F. Beiträge zur Pathologie der Lepra Mutilans, Erlangen, 1867; Obs. I., p. 7.

¹¹ Langhans, Th. Zur Casuistik der Rückenmark's affectionen. Virchow's Archiv., 1875, vol. 64, p. 175.

other sources furnishing the proof of implication of the cord in leprosy. It is true that in the majority of anatomically examined cases the central nervous system was found normal and the peripheral nerves always affected, but on the other hand, the cord has been found to be the seat of congestive and hyperplastic changes, the membranes showing signs of inflammation and the cord itself being found thickened and sclerosed. Tschirjew¹² found the central canal of the cervical region filled with round cells and also atrophy of the cells of the posterior horns and of the columns of Clarke.

Danielssen and Boeck,¹³ in autopsies frequently found a toughening and atrophy of the cord with discoloration of the grey substance and diminution of the ganglia. On the posterior spinal surface meningitis was observed which often implicated the posterior nerve roots. They always found the cord to be more or less diseased. In consideration of these facts therefore, I can understand how in some cases the anatomical change may be in the cord, (perhaps a leprous new formation, with subsequent softening, and final formation of cavities) in others in the peripheral nerves, and in still others in both the nerves and cord. The sharp lines which have thus far been drawn around anæsthetic leprosy as an affection of the peripheral nerves, will, in view of the cases showing partial sensory disorders, have to be modified, or it must be acknowledged that partial sensory disorders as such are not characteristic of syringomyelia.

As well as I believe that the first position, that of considering all the symptoms in anæsthetic leprosy due to a peripheral neuritis, is untenable, so also do I believe that statements regarding the occurrence of partial sensory disturbances as a pathognomonic symptom of central cord affection, have been entirely too categorical, as shown by the following citations from Schultze and Starr.

Schultze¹⁴ writing of the symptoms of syringomyelia,

¹² Tschirjew Archiv. de Physiologie, 1879, p. 615-23.

¹³ Danielssen and Boeck Traité de la Spadalsked ou Elephantiasis des grecs. Paris, 1850.

¹⁴ Schultze. Virchows Archiv. Vol. 102 p. 450.

says: "In multiple peripheral degeneration of the nerves, the peculiar partial sensory paralyses have not yet been observed, particularly the disproportion between sense of touch in contradistinction to sense of pain and temperature." And Starr,¹⁵ writing in 1888, speaking of the differential diagnosis between syringomyelia and neuritis, says: In neuritis all sensations are equally affected, and there is no case on record where pain and temperature sense have been lost with preservation of touch and muscular sense."

About six months ago I instituted a series of examinations of the sensory condition of all cases of peripheral neuritis coming under observation. Of many cases examined only one case showed a partial sensory disorder, and while the form was different from that seen in syringomyelia, it nevertheless will prove of value in supporting my proposition. In this case there was preservation of perception to very low temperature, while all other senses were lost.

The patient was a male, age 46; a brewer, who had his hand and wrist crushed between two kegs of beer.

Open wound, suppuration. Severe degenerative paralysis occurred, implicating the median and ulnar territories. Sensation; anæsthesia and analgesia, particularly marked in the distribution of the ulnar. Sense for temperatures from 40° F. upward lost, but temperatures below that were always felt as cold. A mixture of ice and salt in a test tube, when applied to the skin always produced the same exclamation, "very cold," while a blister could be produced by means of hot water, without causing a feeling of heat or pain. A case very similar to this, has been recently observed by Ziehl¹⁶ and from his article I learn that Nothnagel¹⁷ has reported a case in which after traumatism to the ulnar, reduction of all sensory qualities except that of temperature (heat and cold) occurred. Berger¹⁸ reports a case

¹⁵ Starr, M. A. Amer. Journal of Med. Sciences, 1888, p. 457.

¹⁶ Ziehl Deutsche Med. W'schrift, p. 835, 1889.

¹⁷ Nothnagel. Deutsches Archiv. f. Klin. Med. Vol. II, p. 296.

¹⁸ Berger. Wiener Med. W'schrift, 1872, p. 786.

of peroneal paralysis due to cold, in which the sense to light touch was preserved and localized, while that to pain and temperature was very much reduced. Pick¹⁹ also has lately reported a case of peripheral loss of temperature sense with preservation of all other qualities. These cases, while they do not present precisely the same sensory disorders as those observed in syringomyelia, at any rate plainly controvert the statements that partial sensory disorders do not occur in peripheral affections.

The strange fact that in the peripheral cases of Ziehl and myself, as well as in certain territories in the case of leprosy, sense of cold was preserved while that of heat was lost, can be explained and understood by the interesting and sufficiently well-known investigations of Blix²⁰ and Goldscheider.²¹ In these three cases this occurred in the ulnar distribution, and in all of them there existed a neuritis of the ulnar nerve; it is therefore fair to assume that this symptom is in these cases of purely peripheral origin. The question then arises is there anything in this symptom, preservation of cold sense with loss of heat sense, which is characteristic of peripheral disorder? Is perhaps the diagnostic point between central and peripheral disorders of temperature sense to be sought in this fact?

These questions cannot be answered, for cases of syringomyelia have not been specially examined with very low temperatures, and therefore the sense of cold may still have been present, where now it is supposed to be absent.

My conclusions from these facts and reflections are that:

1. The differential diagnosis between anæsthetic leprosy and syringomyelia cannot always be made.

2. Partial sensory disorders are not characteristic of syringomyelia, but may occur in anæsthetic leprosy as well as in purely peripheral affections.

3. A differential diagnostic point between central and peripheral loss of temperature sense, may lie in its complete loss in the one case and its partial loss in the other.

NOTE.—Since the above was written, an article by Dr. S. M. Suzuki, in the *Sei-I-Kwai Medical Journal*, Tokyo, May, 1889, has come to my notice: Suzuki, in 17 cases of leprosy found the tendon reflexes exaggerated in all of them, and he also believes that this is due to a central cause.

¹⁹ Pick *Wiener Med. W'schrift*, 1888, p. 617.

²⁰ Blix *Zeitschrift für Biologie* xxi, p. 143.

²¹ Goldscheider *Dubois-Reymond's Archiv*, 1885.

ON A CASE OF MULTIPLE NEURITIS AND CEREBRO-SPINAL MENINGITIS.¹

By HENRY S. UPSON, M.D.,
OF CLEVELAND, OHIO.

THE occurrence of multiple neuritis in connection with acute and chronic infectious diseases, has for some time attracted attention, and it seems certain that there is a causative relation between the two; it has been a matter of considerable doubt whether the neuritic process is caused directly by the microbe, or whether it is rather a result of the presence in the circulation, of ptomaines or other poisons produced by bacteria in their growth. The latter view has in its favor the fact that several forms of neuritis are certainly produced by chemical poisons, and that staining and culture tests have as yet given negative results in the few cases of multiple neuritis which been examined in this way. This is, however, by no means conclusive on the point at issue, and in the present fragmentary state of our knowledge of the different kinds of bacteria the appearance under the microscope of the affected tissues may afford considerable information as to the nature of the process.

The following case may in this connection not be without interest.

Mrs. Rolf; age 27. The patient was taken rather suddenly a year ago with violent pains in the arms and legs, followed by paralysis and marked atrophy in all four extremities. She was at that time helpless and unable to feed herself. She was not unconscious, and there seem to have been no cerebral symptoms. The knee jerk is said to have been retained. She recovered from this illness very gradually, and regained considerable power in her arms and legs, so that she was able to be about.

Two weeks ago the patient was taken ill in the same way as before, with violent pains and great prostration,

¹ Read before the American Neurological Association, June, 1889.

some fever. Three or four days ago she passed into a condition of stupor, which has persisted since.

On examination the patient makes unintelligible answers to questions, puts out her tongue feebly when asked to do so, is very quiet unless disturbed. When moved she cries out as if in pain; the neck and all four extremities are tender to pressure. There is opisthotonos which is not very marked. The patient is markedly emaciated, and the muscles of all four extremities are considerably atrophied, especially the thenar muscles and interossei. There seems, however, to be no decided paralysis, as the patient moves both arms and legs occasionally.

There is no facial paralysis; the pupils are equal and react to light; the fundus oculi shows no pathological change, although the optic discs are very pink; the triceps and knee reflexes are quite marked, in fact seem somewhat exaggerated. Electric examination with the faradic current attempted, but unsatisfactory on account of the extreme tenderness of the extremities.

No specific history is obtainable. The patient has never been an habitual drinker.

Provisional diagnosis. Tubercular meningitis of the cord, multiple neuritis a year ago. Examination of the urine for lead recommended.

The stupor continued for two days, when the patient died.

Autopsy twelve hours after death. Lungs congested and œdematous. Two encapsulated masses on right side of vertebral column, adherent to diaphragm, consisting of calcareous matter. Kidneys and liver appear normal.

Brain, dura mater normal, pia much congested over the vertex, with marked serous effusion. Brain substance throughout appears normal. Medulla very firm, otherwise of normal appearance.

Dorsal and lumbar portions of cord and a portion of the ulnar nerve were removed and examined. Pia mater of cord much congested, the veins very tortuous, with serous effusion which has accumulated about the lower part of the cord. On section of the cord and ulnar nerve nothing abnormal was to be seen.

The tissues were hardened in Müller's fluid, and stained with chloride of gold, alum carmine, hæmatoxylin and rosin, and by the Weigert method.

Microscopical examination of the brain, medulla and cerebellum reveals nothing abnormal.

In the ulnar nerve there is increase of the connective tissue elements in almost all parts of the section, and namely a thickening of the septa between the fasciculi, and a loose network between these and the nerve fibres, with interstices which look as if they might have been filled with serum; there is also an increased number of nuclei in the sheaths of Schwan; there are no collections of leucocytes, and nothing to indicate an acute purulent or tubercular process. Carmine and hæmatoxylin staining shows a moderate number of swollen axis cylinders. The myeline sheaths appear many of them swollen and granular.

In the spinal cord there is no thickening of the meninges, the white matter of the cord appears normal throughout, as does also the network of fibres in the gray matter. In the lumbar and sacral portions the nerve-cells of the anterior horns of gray matter are considerably shrunken away from the surrounding tissue, most of them appear to have neither axis cylinder nor protoplasmic processes, in many of them the nuclei are pushed to one side of the cell, so as to form a distinct projection upon the outline of the cell-body; in a few of the cells there are vacuoles, occupying a greater or less part of the protoplasm of the cell. In some of the sections there are in the anterior horns of gray matter, numbers of small bodies of about the size of the so-called corpora mylacia; they have however a more granular appearance than the latter; whether they are portions of degenerated ganglion cells I am unable to say.

In the anterior and posterior nerve roots the myeline sheaths appear many of them swollen and granular, there seems to be little if any increase of the connective tissue.

In the dorsal portion of the cord the anterior horns of gray matter and columns of Clarke appear normal, as does the white matter throughout.

In interpreting the changes described above, there is every probability from the appearances on naked eye examination at the autopsy that the case was one of cerebro-spinal meningitis of the infectious variety; this disease is endemic in Cleveland, and two similar cases chanced to come under my observation within a week of the time at which this case occurred, one of which was verified by post-mortem examination.

The changes in the ulnar nerve indicate an overgrowth of connective tissue, which probably dates from the attack of the preceding year, together with an acute inflammatory condition, evidenced by the swollen axis cylinders, broken down myeline sheaths, and possible accumulation of serum within the neurilemma.

The changes in the nerve-cells are such as have been often described, and doubt has been cast on their significance. That the changes in this case are to some degree pathological, seems to me to be proved by the occurrence of vacuoles, and the pushing to one side of the nuclei; the unusual shrinkage of the cells during hardening, and loss of the cell processes are also significant. Whether these changes are primary, or simply a result of the preceding attack of multiple neuritis, it is hardly possible to decide; the probability is that they are secondary.

No examination of the tissues for bacteria was made.

In reviewing the history of the case in the light of the pathological changes which were found, it seems clear that multiple neuritis was present from the outset; the friends of the patient were positive that on the occasion of the first attack there were from the beginning severe pains and tenderness in the limbs, with little if any stupor or loss of mental power; so that we may conclude that the inflammation of the nerve-trunks was coincident with, if it did not precede, the meningitis.

The appearances in the ulnar nerve do not correspond with those described as occurring either in ordinary Wallerian degeneration, or in the usual forms of multiple neuritis; aside from the thickening of the neurilemma, the changes would all be very readily explained by supposing

congestion and a serous exudate of sufficient severity to cause pressure on, and consequent changes in some of the nerve tubes; the membranes of the nerve trunks, usually called the neurilemma, seem to have been primarily affected, and this with the very early appearance of the neuritis tends strongly to show that the inflammation was directly infectious in origin, rather than an indirect result of the meningitis.

The occurrence of multiple neuritis with cerebro-spinal meningitis was first suggested by Mills, of Philadelphia, in 1888, although unfortunately in his cases he could obtain no autopsy. He found in the acute stage, as in this case, symptoms of an irritative rather than of paralytic lesion. This is readily understood if we suppose the process to be identical in nature with that in the pia mater of the cord and brain.

In the epidemics of multiple neuritis which have been described, it is probable, as has been pointed out by Eisenlohr, that the specific cause is the same as that for cerebro-spinal meningitis, but definite knowledge on this point can only be furnished by further investigation, especially in the line of bacteriological research.

THE LATER HISTORY OF A CASE OF FOCAL EPILEPSY FOR WHICH TREPHINING AND EXCISION OF THE MOTOR CENTRES WERE PERFORMED.¹

By JAMES HENDRIE LLOYD, M. D.,

Visiting Physician to the Nervous and Insane Department of the Philadelphia Hospital.

AT the meeting of this association held in Washington, in September last, I reported with Dr. John B. Deaver, a case of focal epilepsy which had been observed and studied in my wards in the Philadelphia Hospital, and in which Dr. Deaver had operated. The case was reported² as successfully treated by trephining and excision of the motor centres. At the time of this report three months had elapsed without the patient having suffered a return of his convulsions, although before the operation he had had daily a series of epileptic attacks numbering from fifteen to twenty-eight. In the discussion of this case, which followed the reading of the report, Dr. David Ferrier, of London, said, in effect, "that while such cases were of interest and value it was, as yet, too soon to claim that the case was permanently cured." He referred to a case of his own in which a similar operation had very much diminished at first the number of epileptic seizures but had not permanently abolished them. In closing this discussion I promised to report the subsequent history of our case if the patient's convulsions returned, and in accordance with this promise I have prepared the following notes :

It may be well to refer briefly to the symptoms in this case and to the operative procedures. The patient was a man, aged about 35, who had had epileptic fits for fourteen years. These fits were ushered in by a sensory aura running from

¹ Read at the meeting of the Am. Neu. Ass'n, Long Branch, June, 1889

² American Jour. Med. Sciences, November, 1888.

the index finger of the left hand. The signal motor symptoms were twitching of that finger, rapidly followed by convulsive movements in the left arm and in the left face. These parts were sometimes alone involved, with slight, if any, loss of consciousness; while at other times the fit became almost general, but always worse on the left side, with greater loss of consciousness. In the intervals between the attacks the fore and middle finger and the muscles of the left face were quite perceptibly paretic. The operation consisted in exposing the middle and lower portions of the ascending frontal and parietal convolutions and in removing the portions of these convolutions which were found by direct faradic stimulation to preside over flexion of the fingers and contraction of the face muscles, no other method being adopted to find these but the use of faradism.

After the operation the patient had no convulsive seizures, except a very few minor attacks during the first few days, up to the time when the report was made—a period of three months. Paresis of the left fore and middle finger and of the left face persisted.

A few weeks after this date the patient was reported by the attendants to have had an epileptic seizure. He confirmed this report himself. From that time up to his departure from the hospital, six months later, he had ten more seizures. The character of these attacks, as far as I could make it out from the reports of attendants and the patient himself, was very similar to what it had been before the operation; the sensory aura and the rather localized nature of the fits, with only partial loss of consciousness, being apparently the same. These return seizures were nearly always nocturnal, so they were not observed by any person trained in observation. The chief and resident physicians never had an opportunity to observe them—so the record rests entirely upon unskilled, but apparently authentic, observation.

The patient suddenly left the hospital and has not been heard of since. He was seen once subsequently by the writer, riding a horse bare-back on Market Street, West Philadelphia—having evidently secured employment at a training and sale stable.

As a result of this operation we have, briefly, a suspension of all epileptic seizures for almost four months; then a return of them at rare intervals up to nine months, when the patient disappears from view. The total number of return seizures during the five or six months of their observation being about ten, which is less than the patient had had often in one day before operative interference.

It is disappointing, however, to have to record the return of any fits whatever and however few, for if this operation, which is based upon such interesting and valuable physiological work, is to endure as a recognized surgical procedure it is obvious that it must furnish results which are complete and beyond criticism. The return of any convulsive symptoms whatever exposes the operation to the suspicion that something more remains to be done, or to the condemnation that nothing more can be done to make the excision a success.

The case suggests to my mind a few ideas which I will state briefly.

Faradic stimulation was relied upon almost entirely to map out the centres presiding over the movements of the face and arm. I noted at the time, and stated so in my report, that some areas of the cortex in immediate contiguity to the areas excised were not excitable at all, at least did not give any muscular response anywhere. In commenting upon this fact in his discussion of Dr. Mills' paper before the Congress, Mr. Horsley said that the substances used in antiseptic surgery—such as the corrosive sublimate—had an effect in diminishing the excitability of the brain cortex to faradic stimulation. At a subsequent operation upon a child, the patient of Drs. Mills and Roberts, this fact appeared to be illustrated. I made the faradic exploration, by the kindness of these physicians, and although a strong current was employed no response anywhere was elicited. The area exposed and faradised was the region presiding over protraction, and also the upper regions of the ascending frontal and parietal convolutions. The patient suffered with epileptic attacks, ushered in by a very marked and characteristic movement of protraction of the arm.

Cortical epilepsy without gross lesion can of course be localized by a strict observance of the principles of cortical localization only as illustrated in the experimental work of the brain physiologists. But these principles are not as yet complete and unalterable, for even the most advanced and enthusiastic of these pioneers will not claim that the science is as yet in more than its early stages of development. It is not therefore strange that surgery in its practical application of this, as yet incomplete, knowledge should not have been able to attain a precision of diagnosis or to elaborate a technique which are infallible. I think these cases, therefore, are of great value for what they teach of success and what they demonstrate of failure. The great desideratum is a more precise knowledge of the limits of the various centres, or special areas, and a method of demarkating them more exact than has been as yet suggested. I think the one defect in our case, and most probably in all cases yet operated upon, was the fact that with the greatest care it was impossible to say that *all* cortical tissue had been removed which presided over the affected muscles. This difficulty is due to several causes. First: it is not easy to recognize definite small areas of brain cortex through a small trephine wound. Second: when recognized it is not easy to define their exact limits; and third, the diffusion of the faradic current to neighboring centres, and the sedative action of antiseptic substances, increase this difficulty still more. Again, the healing process, even under aseptic conditions without the formation of pus and undue adventitious products, may leave a contracting, hyperplastic and irritating scar which possibly causes a return of convulsive seizures after an interval of exemption. This element of mischief has not been dwelt upon with the emphasis it seems to demand. If it is a cause of return spasms, it is difficult to determine how it can be avoided. Frank has claimed that a distinction exists between the spasm produced by irritation of the cortex and of the descending fibres below—stating that in the cortex alone have we the true clonic, epileptiform phenomena, while irritation of the motor-descending fibres produces a spastic reaction. If this is

always so the scar tissue forming after an operation on the cortex involving the white substance beneath, would, it seems, produce a tonic, and not a clonic fit. In our case, however, the return spasms were clonic, and apparently precisely similar to the original attacks. The conclusion would be, that not scar tissue, but unremoved cortex, was the cause of the return. The similarity of the return-seizures has been observed, I believe, by others.

A PARASITE OF A BIRD'S BRAIN.

By W. C. CAHALL, M. D.,
Philadelphia, Pa.

THE functions and diseases of the brain of a bird, in the strictest sense, belong to the comparative anatomist ; but a brain, even though it be a bird's, which tolerates, without apparent harm, a nest of parasites upon and within its substance, cannot fail to have great suggestive interest to the physician, and especially the neurologist.

The *Plotus anhinga*, or Snake-bird, as it is popularly known, is found in the Southern Atlantic and Gulf States. Its habitat is along the rivers, lakes and lagoons, in the waters of which it finds its food. It is of the cormorant family, and an expert fisher, being able to go a long distance under water. Its food, according to Audubon, consists of small fishes, insects, eggs of frogs, crawfish, young alligators, and small water-snakes.

During the winter of 1867, Dr. Jeffries Wyman, of Harvard University, spent the months of February and March on the St. John's River in Florida. In dissecting the snake-bird, he discovered, in seventeen specimens out of nineteen examined, a mass of threadworms, varying from two to fifteen in number, lying between the cerebellum and the skull, and within the folds of the arachnoid and the pia mater.

He found also in the gizzard and stomach a large number of parasites. He says: "While some of them simply adhered to the mucous membrane, others had their heads thrust deeply in, or their bodies were almost concealed by being buried in the gastric follicles. They are about seventeen millimeters in length, and their oviducts contained an abundance of eggs."

Dr. Wyman reported the results of his investigations to the Boston Society of Natural History in October, 1868.

In 1874 Prof. J. W. P. Jenks, of Brown University made

an extended tour through the then wild region of southern Florida, and according to a promise given to Dr. Wyman, made an elaborate search for the parasites in the brain of all the *anhinga* he could shoot. The material thus gathered was presented to Dr. Wyman, who intended to incorporate it in a fuller report the ensuing Fall, but his sudden

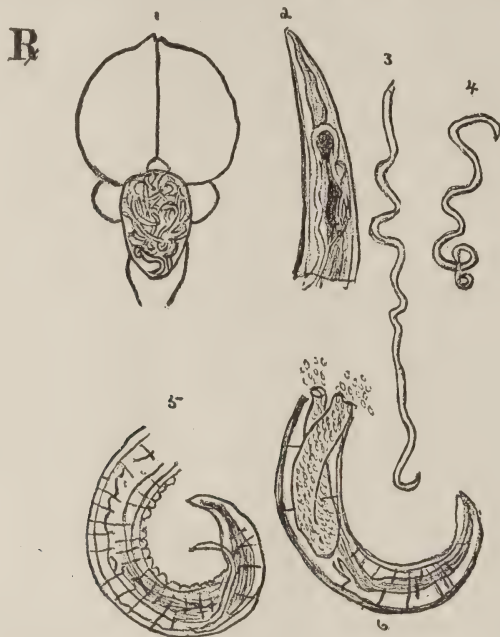


FIG. 1.—Showing relative position of nest of parasites to that of the brain.
 FIG. 2.—Head of worm (magnified).
 FIGS. 3 and 4.—Female and male thread-worms, twice their natural size.
 FIG. 5.—Posterior extremity of male worm, (magnified).
 FIG. 6.—Posterior extremity of female worm, showing oviduct distended with eggs, (magnified).

[After Wyman.]

death in September of that year prevented him from carrying out his promise. The report was never made, and so far as I have searched, the report of Dr. Wyman in 1868, and a short note in Prof. Jenks' *Zöology*, comprise all the literature upon the subject published.

But in a letter of Dec. 1st, 1888, Prof. Jenks has very kindly given to me the results of his investigations. He writes :

"As I left Dr. Wyman he enjoined upon me to save all

the brains of the snake-bird I could, and verify or disprove his paper above alluded to. In that paper Prof. Wyman did not attempt any explanation, but simply stated the fact that at least nine out of every ten of the adult *anhinga* had a bunch of parasitic worms, about an inch in length, and to the number of fifteen more or less, coiled together on the top of the brain. As I found them by the hundred in the different rookeries I visited, I not only verified his statement, but also by taking them in all stages of growth, and carefully examining scores of them, I found the half-fledged had no parasitic worm upon the brain, but a few in the stomach, and in those more fully fledged, at different points in the muscles, as though making their way to the brain."

This is a very important observation of Prof. Jenks, and may be found to be the true explanation of how the parasites find their way to the brain. Be this as it may, in the adult bird the parasite is found in no other part of the body except the brain.

Wishing to satisfy myself by a personal examination, I have received from Prof. Jenks the heads of several of these birds and have made a careful study of them. Upon removing the top of the skull, I was struck by the constant appearance of an intensely congested spot of the brain membranes of about half an inch in length and a quarter of an inch in breadth, and occupying a uniform position, so uniform that the skull could be trepanned and the parasites successfully extracted from the living bird.

This position is the central line of the upper surface of the cerebellum, and beginning at the fissure which separates the cerebellum from the cerebrum.

Within this circumscribed area the several thread-worms were found rolled up into a loose ball, and while they could scarcely be described as encysted, still they appeared to be in a condition of harmless inactivity.

The microscope revealed the nature of the parasite to be that of a thread-worm. Both sexes are usually found present in the same nest. They vary from three quarters to one and half inches in length. The female, which is the larger, is frequently found with its oviduct distended with

an enormous number of eggs and hatched young. In view of this great reproductive power of the thread-worm, it becomes an interesting question as to what becomes of this constantly increasing brood, since no more than twelve or fifteen adult worms have been found in the brain at one time.

Dr. Wyman, reasoning from analogy, supposes them to be of a migratory kind, which pass a part of their life in another animal or bird. But this supposition, and it is very probably a correct one, offers no explanation of the manner in which these young parasites find their way out to their future host, for the parasite is never found in any other part or tissue of the bird than the one within the cranium.

Prof. Jenks found repeated evidence of their progress from the stomach to the brain in the half-fledged bird, but never in the adult any similar migration outward.

The most noteworthy feature about this disease is the remarkable tolerance of the brain to the mass of foreign matter compressing its surface. So large is this mass, and so great its pressure that frequently the natural outlines of the cerebellum have been lost owing to the hollow upon its upper surface made by the growing thread-worms.

Küchenmeister, in his "Manual of Animal and Vegetable Parasites," gives a plate illustrating the brain of a sheep suffering from the "staggers."

This intractable disease is due to the presence in the brain or its membranes of a minute cysticercus, sometimes but one, seldom more than three or four, which after wandering around, finally becomes imbedded in or upon the brain mass, and which gives rise to a marked localized inflammation of the structure surrounding it. In the section on Tumors of the Brain and its Envelopes, of Pepper's System of Medicine, Drs. Mills and Lloyd have collected some cases of direct bearing upon the present subject.

After eliminating all the cases of tumor of parts of the brain other than the cerebellum, and also all those which are situate *within* the cerebellar structure, producing by their presence lesions of the organ, there remain three cases, Nos. 73, 74 and 75 of the table, which present points of similarity to the parasitic tumor of the snake-birds brain.

In each of these three cases the tumor was confined to the envelopes of the cerebellum, and the symptoms which finally led to the death of the patients, being produced solely by the mechanical pressure of the foreign growth. The most characteristic symptoms of tumor of the cerebellum were found to be occipital headache, incoordination of muscles, a tremor of limbs, and a staggering gait.

In case 74 the tumor was the size of a hen's egg and lodged between the superior surface of the cerebellum and cerebrum. The most prominent features noticed in this case were violent headaches, gradual loss of sight, anæsthesia and paralysis of left arm and leg, intellect dull, nausea, hiccough, asphyxia by strangulation.

In case 75 the tumor was of the size of a green gage plum, growing from the dura mater of the left side, and by its pressure produced a deep depression in the left lobe of the cerebellum. In this case there were, as in case 74, headache, frequent vomiting, stupor, but, unlike it, was not blind, and had the power to move arms and legs, although the patient could not stand. Died in a convulsion, the right side being the most affected. Thus we find that marked nervous phenomena invariably accompany the presence of the comparatively insignificant cysticercus in the sheep's brain, and equally alarming symptoms are produced by a small tumor, pressing upon the cerebellum of a man, while nineteen out of every twenty snake-birds carry about in their skulls a mass of thread-worms, which, in comparative bulk, would be equal to a handful of large sized angle or fishing worms resting upon a man's brain, and this is done without any apparent disturbance of the functions of the bird, for the snake-bird is the equal of any of the cormorant family in their success in fishing, in their wariness of the hunter, their skill in building their nests and care of their young, and all other social duties which a bird is called upon to perform.

Why is the brain of a bird different from that of a sheep or of a man?

This is one of the instances where reasoning from analogy, based upon comparative anatomy or pathology, would be misleading.

A CASE OF LATERAL HEMIANOPSIA, WITH AUTOPSY.

By WILLIAM NOYES, M.D.,

Second Assistant Physician, Bloomingdale Asylum, New York.

From the Clinical Records of the Bloomingdale Asylum, New York.

THE following case is of interest from the fact that the autopsy confirmed the diagnosis made before death, by Dr. E. C. Seguin, as reported by him in his "Clinical Study of Lateral Heminanopsia," in the JOURNAL OF NERVOUS AND MENTAL DISEASE for August, 1886. For the sake of completeness in the record Dr. Seguin's report of the case (p. 448) is again given here :

"GROUP II.—Cases of bilateral hemianopsia without hemiplegic symptoms. Lesion probably in one cuneus and adjacent gray matter.

"CASE I.—Male, æt. 52. Seen in consultation with Dr. H. H. Tinker, June 13th, 1882. Former health good, with exception of severe attack of migraine with vomiting frequently in the last ten years. Has had rheumatic manifestations, but positively denies any venereal disease.

"On February 23d, 1882, in a severe attack of migraine, after violent vomiting, suddenly found that he could not see on his left. Later in the day was completely blind. No paralytic phenomena or unconciousness; for a week or ten days was ill with slight fever (100-102); after this, sight improved, but blindness to left remained.

"On April 20th, in the midst of a debate, without loss of conciousness, or paralysis, suddenly found himself 'mixing up his words.' Was able to write at once after attack. When seen later on same day by Dr. Tinker, there was slight hemiparesis. Speech has since greatly improved.

"*Examination.*—The only trace of right-sided paresis is a slight hanging of the lower part of the face; grasp normal; no hemianæsthesia; speech good. Pupils of the eye and

muscles normal; no lesion of fundus; with glasses for presbyopia reads easily; has typical lateral hemianopsia to left, with a concentric limitation of nasal half field of left eye. Heart large, but without murmur. Albumen in each of three samples of urine.

“Pathological Diagnosis.—Two separate attacks of cerebral hemorrhage: 1st, with destructive clot in right cuneus, and a slight ecchymosis (?) in the left cuneus, (total obscuration of fields for a few days). 2d, clot in or near the speech centre of Broca in the left hemisphere.”

The patient entered Bloomingdale Asylum December 1, 1885. The history of the case subsequent to the record given by Dr. Seguin, is as follows, the facts being obtained at time of his admission, from a letter from a brother of the patient: “. . . He was examined in June, 1882, by Dr. Seguin, who said that with rest for some months there was every prospect that he would so far recover as to be able to resume business in some moderate way, though Dr. S. said there had undoubtedly been a hemorrhage in the brain During the winter, 1882-3, he did transact some business. In April, 1883, while in a business office, he again suddenly found he could not speak. He went home and found when there that he had partially lost the use of his right arm. His speech was so thick as to be nearly unintelligible—very much worse than at present. From this also he rallied, but not completely. His speech has never returned altogether and his gait became somewhat feeble. But there was no mental failure that I could detect. He could discuss business matters clearly and well. There was perhaps some irritability and unreasonableness, but as to this there is no absolute certainty. He read a great deal and with enjoyment, and enjoyed travel.

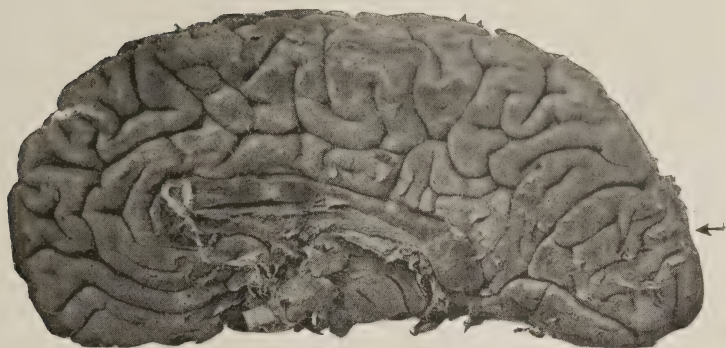
“On Thanksgiving Day (last week in November) 1884, he had a convulsion and was unconscious for some minutes. He had another some months after. From this time mental failure was observed, forgetfulness, irritability, persistence in unreasonable wishes and plans, etc. Still, up to May, 1885, he could listen intelligently to conversation and take part in it, enjoyed being read to, and remembered

past events. Since then the failure has been rapid. He has had another slight convulsion some months ago. At present his mental condition varies much at different times. He knows his friends, and even acquaintances, but forgets the events of each day. He is helpless in many ways, cannot feed himself nor attend to the calls of nature properly without assistance."

On admission to the Asylum he was quite feeble, walking with much difficulty and dragging his right foot painfully. The right arm was in a state of contracture; tongue tremulous and deviated to the right; pupils reacted normally, the right being possibly a little larger than the left; heart sounds normal. His mental symptoms presented nothing worthy of note, being simply those of dementia, secondary to cerebral hemorrhage. He was entirely unable to give any statement of his condition, simply answering "yes, yes" to every question.

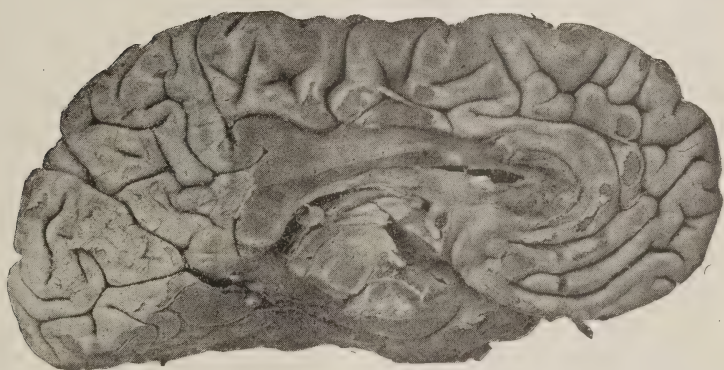
Mental and physical failure were rapid, and he died of exhaustion May 22d, 1886. The brain was removed the same day. The accompanying photographs of the two hemispheres were taken after the brain had been thoroughly hardened in Müllers fluid. The lesion in the right cuneus shows very plainly, the convolutions having undergone considerable atrophy. The photographs are on the same scale, thus showing that the right hemisphere, as a whole, had become smaller than the left. There is no visible lesion in the left cuneus, the convolutions retaining their normal size; the roughened surface in the photograph being due to an accidental abrasion of the hardened brain. No examination of the internal parts of the brain was made, as it was thought best to submit it to still further hardening in the hope of discovering any degeneration there might be in the optic tracts.

Macroscopic section of the right hemisphere showed no evidences of degeneration of the optic tracts visible to the naked eye.



RIGHT HEMISPHERE.

The arrow shows the lesion in the cuneus, with the marked atrophy and wasting of the convolutions.



LEFT HEMISPHERE.

The convolutions in the cuneus are shown to be full and of undiminished size.

UNUSUAL EPILEPTIC PHENOMENA.

By C. M. HAY, M.D.,

Asst. Physician State Asylum, Morristown, N. J. Formerly Resident Physician, University Hospital, Philadelphia, Pa.

AN epileptic aura is not, I am convinced, as frequent an accompaniment of the paroxysm in its various degrees as text books, (especially English authorities) would lead us to expect. The number of cases in which it is absent has been a matter of surprise to me, while its complex nature in certain rare cases is equally surprising. Having observed with much interest some unusual, and even unique pre-convulsive phenomena and also some unusual cases of the disease, an account of some of these may be interesting to the profession.

In my service at this institution there are 57 epileptic patients, and, with the view of determining the relative frequency of the aura in these cases, I carefully examined 33 of them, whose mental condition admitted of reliable results. Of these, 20 were male, and the remainder female patients. In 16 of these cases (or 48.5 per cent.) there was no distinct aura of any kind, while in 17, various sensations were described. In 5 of the latter cases, the aura was gastric in origin, and went rapidly to the head. The sensation was variously described as a painful (in one case), burning, or distressed feeling. In three of the 17 cases, it was characterized as a general bodily sensation, or universal aura, in one being a "coldness all over," and, in the remaining two, a sensation of numbness, combined, in one, with dancing colored lights before the eyes. In three cases, the aura was confined to the head, and consisted of a sharp pain (in one case), vertigo in the second, and a loud noise in the left ear in the third patient. In two cases, the warning note of the paroxysm consisted of a "distress," and a sense of constriction respectively, beginning within the thorax, and

going to the head before they became unconscious. In only one case did the aura start in an extremity. In this case the paroxysm began with numbness and tingling in both feet usually, (sometimes one), and this gradually goes up the legs to the knees, increasing in intensity. When the aura reaches the knees, the patient becomes unconscious, and states that it never extends beyond that point. The remaining three of these 17 patients exhibited more complex sensations before their convulsions. The first of these before each of his seizures, which usually occur once a month, has visions of various animals float before his eyes. The most constant he describes as "large, heavy birds," although horses, cows, dogs, and other animals enter at times into them. On two occasions it has seemed to me that his ordinary convulsive attack has been substituted by an excessive amount of these visions. On these two occasions he described the visions with unusual distinctness. They were greatly prolonged, and were followed by no convulsive seizure. Usually, however, an hallucination of sight is the immediate precursor of his epileptic attacks. The second patient presents a more complex aura. It consists of sensory and special sense parts, the latter being, as in the first case, a hallucination of sight. His paroxysm is ushered in by a sudden numbness and tingling in the hands and feet, with a sensation of vertigo in the head. Succeeding this, a momentary blackness comes over the sight, followed by a play of colors, which quickly arrange themselves into a picture, which the patient describes minutely. This consists of a central figure of an old man, clad in flowing garments, with long white waving hair, walking along the sea shore, and behind him is a boat with men in it. The shore is of bright scarlet, while the sea, boat and figures are of natural colors. This vision is a constant one, and occurs before each convulsion. In some of his attacks, the boat with its occupants are absent from the picture, but the central figure of the old man, and the other surroundings, as described, are always present. The third patient has still more complex warnings before her paroxysms, in which ordinary sensation and two special senses, (sight and

hearing) are involved. Immediately preceding a convulsion she has a vision of angels, surrounded by a dazzling light, and hears voices singing. Preceding this, there is a buzzing in the ears, and a pain flashes through the head. During the time she sees the vision she automatically repeats a certain sacred verse, (always the same one), and then unconsciousness and the convulsive phenomena follow regularly. I have frequently seen this patient start up from her bed in the evening and commence repeating this verse, but, when half through with it, it would be cut short by a loud scream and the ordinary sequelæ in such patients. Afterwards she has repeatedly told me of this same vision, and it appears to be a constant accompaniment of her paroxysms.

Since writing the above, I have found in another patient curious prodromata. The patient is a male aged 20, and has had epilepsy since his childhood. Almost constantly before an attack he fancies himself in some familiar place, other than where he really is. Sometimes it is at the home of his boyhood, while, at other times, it merely refers to an adjoining apartment, in which he has previously been seated. Before other attacks, he has a brilliant play of colors before his eyes, which commence as broad bands of bright colors, running in various directions, and then begin to whirl about until unconsciousness ensues. In numerous attacks these prodromes are entirely substituted by a perversion of sight, which takes the form of a mouse running over the floor towards him. When the animal reaches him, he loses consciousness. He has had a few convulsions without an aura of any kind preceding. It is interesting here to note that this patient has always had a horror of a mouse.

The epileptic convulsion itself is subject to so many modifications in different cases, that rarely do we meet with cases that can be designated as very unusual. I think, however, that the following cases deserve attention, as being very unusual cases of the disease. These have been under observation for a number of years, and their previous histories are obtained from the records of this house.

CASE I.—Mrs. A. W., age 27; no history of any hereditary taint. At the age of five she began have to epileptic convulsions of the ordinary type. Her disease progressed very rapidly, so that, when 9 years old, she was sent to an institution for treatment, her convulsions being so frequent that it was impossible to properly care for her at home. She remained under treatment for two years, during the first of which it is stated that frequently she would have as many as thirty-six convulsions a week. At the end of two years however, she was discharged, greatly improved. During the two years following her discharge she had no seizures, and the child improved mentally and physically to a marked degree. Then her epileptic seizures again, though slowly, recurred, but it was now noted that their character was different from her previous attacks. This difference consisted in a tendency to certain violent acts, preceding the attacks. These acts varied with the surroundings, and generally consisted in her flinging in any direction any article which happened to be in her hands, or in overturning movable furniture. Succeeding these acts she would have an ordinary convulsion, in which she fell to the floor, and had tonic and clonic general spasms. Later on, a further alteration occurred, and she began to run a variable distance before her paroxysms. At first she would run only a short distance, and gradually this was prolonged, until she would frequently run to a neighbors house and have a convulsion, afterwards remembering nothing of such an act, and wondering how she came to be there. This feature again made it necessary to remove her from home, and she was first admitted to this institution December 3d, 1879.

On admission, the patient was in good physical condition. She could read and write well, and her mental condition, as evidenced by her conversation showed great improvement since her discharge from the institution to which she was first sent. Her two years of immunity from the disease had allowed her faculties to develop, and no one would have classified her as "weak minded." She could give no account of her attacks, and only knew of their occurrence when informed afterwards that she had

had them. Under treatment she improved again; her seizures grew less frequent, while their character remained the same. She was discharged greatly improved, July 31st, 1882. She remained at home, having very infrequent and mild convulsions until February 19th, 1884, when she was readmitted on account of an exacerbation of her disease. During the time she remained at home she married (upon the advice of her mother, who hoped that the marriage state would be beneficial to her), and had one child, after the birth of which her attacks increased in frequency. During 1885-1887 she continued to have her paroxysms at intervals of from ten days to several weeks, with the exception of several longer intermissions, one of which began October, 1886, and lasted a few months. During the last two years a further evolution has taken place in the manifestations of her disease. This consists in the addition of pure running attacks of the duration of the convulsion and frequently substituting one. Gradually her convulsions became less frequent, and these running attacks more frequent, until the latter outnumbered the former, and have at present become by far the most frequent expression of her epilepsy. Her ordinary attack may now be described as follows: Suddenly starting from any employment at which she may be engaged, the patient utters a piercing scream, and begins to run wildly up and down the room she may be in, with wide open, staring eyes and dilated pupils. Any obstruction is overcome, and, while in this state, the woman is a marvel of strength; almost incredible stories being related by her attendants of her prowess. On one occasion she utterly demolished a partition of strong boards, and, on another, she wrenched away a settee on which a number of persons were sitting, and then dashed it down in a wreck and continued her flight. During this time she emits a low moaning noise. She then, after a variable period of from a few seconds to several minutes, ceases running and becomes quiet. She then instantly begins to arrange in order all that she has disordered during the spell. As this occurs most frequently in her own room, this feature is best appreciated there. She here gradually restores to order an

apartment that has literally been torn to pieces, almost every article being upset, including the bed, with the clothes. During this time her manner is abstracted and the pupils dilated. She answers questions slowly and correctly, but never essays to speak, unless questioned. The face is usually pale. It is not until everything has been returned to its place, even to minute toilet articles, that consciousness returns, and she then remembers nothing of the attack, and indeed would not know of its occurrence unless informed. At rare intervals she has a mixed attack, in which she runs, falls, and is convulsed, and then immediately gets up and rearranges her room as in a pure running attack. The remarkable feature of this case is that if the patient is held at the beginning of the attack, it instantly assumes the nature of an ordinary epileptic seizure. Between the convulsions, the patient is a good looking female, of exceptionally fine physique, and shows, as yet, little mental deterioration, notwithstanding the long duration of the disease.

CASE II.—The second case resembles the first, and is as follows: Mary G., age twenty-five, single; family history shows predisposition to nervous disease. There is no history of syphilis or tuberculosis. She had scarlet fever at the age of two and recovered, "without good sense." At six, she began to have fits, and a few months later a tape worm was expelled. Unfortunately (as in a few cases already reported), her convulsions did not then cease, but continued at irregular intervals, gradually growing more frequent, until, when fourteen years of age, her mental condition began to deteriorate, and she became passionate and destructive. She was admitted here first on September 24th, 1880. Under a course of bromides she improved considerably, and was discharged from the institution, July 31st, 1881. She remained at home, having occasional convulsions, until July 15th, 1886, when she was readmitted here on account of a change for the worse in her mental condition, maniacal attacks being added to her previous symptoms. Her physical health was even improved at this

time. Since her last admission this patient has had running attacks develop gradually, as in the first case. They are of the same character, more frequent, and they nearly wholly substitute her ordinary epileptiform convulsions. It would seem in this case also, as in the first, that the type of her seizure has undergone a gradual evolution. The last seizure in which she became convulsed occurred about three months ago, while, since then, she has had almost daily running seizures. Her mental condition is now advancing rapidly to complete dementia. The patient remembers nothing that occurs during her attacks, but they are not followed by epileptic automatism as in Case I. The third case, in which consciousness is not lost in all the attacks, is as follows :

CASE III.—Maggie S., age twenty-four, single ; no history of any nervous disease, syphilis, or tuberculosis in her family. Her physician gave her history as follows : At the age of two she began to have chorea. This began gradually, and increased to a violent form of the disease, continuing until her seventh year, when it ceased. During the following year, she began to have epileptic convulsions at long intervals, and continued to have them until March, 1887, when they ceased. Shortly after her last convulsion she began to have attacks of maniacal excitement, and was admitted here, March 28th, 1888. She had had no convulsions since March, 1887, but had had, at intervals corresponding to her convulsive attacks, periods of acute mania. From the history obtained from her physician, it would appear that all her former attacks resembled ordinary epilepsy, but since her admission here, peculiar seizures have been noted in addition by several of the resident medical staff, and I have been fortunate enough to have witnessed several of them. Generally she has some warning of their approach by sharp pains in the lower extremities, with a sense of dizziness in the head, and, at times, gastric distress. She then finds a seat, usually placing her hands upon her knees. By this time a fine twitching occurs in the muscles of expression ; the eyes look straight for-

ward; the pupils are equally dilated; but the woman is conscious, answering questions correctly and expresses her sensations. This is succeeded by quick tonic and clonic spasms of the spinal muscles, especially in the cervical and dorsal regions, so that with each one the head is slightly thrown back, and the hands involuntarily lifted from the knees. At times the spasms involve the extensor muscles of the arms and legs, so that the legs are straightened, and the arms are drawn at right angles to the body. The contractions occur several times in quick succession, and then an interval of a few seconds occurs, to be succeeded by another series of contractions. During this time the woman answers questions, and, during the spasms, utters various exclamations, such as a person might do when receiving sharp shocks from an electric machine. The pulse during the attack is small and frequent, and the seizure itself frequently lasts as long as fifteen minutes. In some of these attacks the patient momentarily loses consciousness. About once every three months this patient has an ordinary attack of grand mal, typical in all respects. Treatment in this case has not seemed to influence the disease; while the patient enjoys good physical health and has had no return of maniacal symptoms during the past year. In this case the existence of chorea prior to the commencement of epilepsy is worthy of note.

The last anomalous case among these 57 patients is one who has both hysterical and epileptic convulsions, and they frequently substitute each other. Her attacks are typical of either hysteria or epilepsy, and never assume the hysterio-epileptiform type. Her history is briefly as follows:

Mrs. T., age 36; has been subject to epilepsy from the age of five years. On admission, one year ago, she was having, on an average, four convulsions a week. She was naturally of a nervous disposition, but her family history could not be further obtained. Since admission she has improved, and her attacks of epilepsy are now comparatively infrequent. I have known her to go for two months without any epileptic seizure, but, during that time, she would have several characteristic hysterical convulsions.

During these, opisthotomos frequently developed and the convulsions were unusually severe and characteristic. One that occurred on May 26th, 1889, was as follows: While in conversation, she complained of feeling "very nervous," and said that she was "going to have a spell." In a few moments, with an exclamation, she fell off her chair on the floor, being careful not to hurt herself, globus hystericus occurred, and for some minutes she lay there quietly. Then rigidity developed, commencing first in the arms, and extending over the whole body. A series of postures were now assumed with lightning rapidity by the patient, and she then became quiet. Examination showed universal cutaneous anæsthesia extending over the conjunctivæ. After this interval, rigidity recurred and was succeeded by quick successive attacks of opisthotomos, the patient assuming first that position, then another, then reassuming the first with extreme rapidity. At this time the arms were rhythmically waved, and, at the close of the attack, were extended at right angles to the body, and the face took on an ecstatic expression not common to the disease in this country. After two hours, the patient recovered, and had another similar attack the same evening. Her epileptic seizures are wholly without warning of their approach, and are typical of the disease in all respects. The patient herself readily differentiates between the two kinds of paroxysms.

The change of nature which the epileptic attack has undergone in the first three cases is certainly remarkable. In the first two cases the paroxysms have been nearly similarly modified—in both the running attacks have gradually developed and substituted the ordinary convulsions, until the former have become the rule and the latter the rare exception.

In this first case, the manner in which the seizure is recovered from, consciousness being absent, while the woman automatically answers questions sharply put, and re-arranges her room perfectly, seems to me to present a very unusual and complex case of this curious mental condition. While in this state of epileptic mental automatism the pa-

tient resembles a somnambulist. At times she regains consciousness more rapidly than at others, the change from one state to the other being almost immediate and complete, careful examination failing to elicit any consciousness on the part of the patient of her re-arranging her room during the preceding half hour. Another interesting feature of this condition of automatic consciousness is the ability of the patient to perform certain suggested acts, and afterwards have no remembrance of doing them. On two occasions I have thus made the patient assume various postures, and return to bed, arranging the clothing as directed, while on the following morning she had no recollection of my visit to her room. Cases of epileptic automatism have been reported by many writers, including Bucknill and Tuke (4th edition, page 337 of "Psychological Medicine"), H. C. Wood ("Nervous Diseases"), Hammond ("Treatise on Insanity"), Legrand du Saulle ("Etude Medico-legale sur les epileptiques," Paris, 1877), and many others; but I have not seen the report of any case that combines, as this patient does, pure running attacks with such well marked and persistent epileptic mental automatism. Automatic procedures after ordinary epileptic convulsion are not uncommon, and several of my patients exhibit various degrees of it. One always undresses immediately after an attack, and this is said by Gowers to be a frequent performance in such cases. The great similarity between this mental state and double consciousness is referred to by H. C. Wood ("Nervous Diseases," page 460). He also details a case (page 106), in which (as in Case III.) consciousness was retained during the attack, which commenced always with an aura in the hand, extending to the neck and ended with convulsive movements of the muscles below the point to which the aura attained. The third of the preceding cases seems to me to present a rare form of epileptic seizure, inasmuch as in some of the attacks, as already described, consciousness is not lost. This was determined both by inspection of the patient during the paroxysm, and also by careful questioning. The patient, who is an intelligent lady, after such attacks can

accurately give all the events that occurred in natural order, including conversation that was carried on during the attack; so that it may safely be concluded that at no time in such of her attacks was consciousness impaired or lost. The occurrence of sharp pains in the lower extremities, associated with gastric uneasiness and vertigo preceding the attack, together with the convulsive twitching of the facial muscles, and the intermittent tonic and clonic contractions of the other muscles as already referred to, give additional interest to this form of epileptic seizure. Its long duration and the comparatively slight after-effects (headache and malaise) are also worthy of note. When loss of consciousness does occur in these attacks it is momentary, but it may occur several times during one attack. The patient never falls except in her ordinary epilepsy, which occurs, as already stated, about once every three months. These symptoms would point to the existence of some local organic lesion, while the history of the case, with the absence of other signs, indicates that it is more probably one of the idiopathic forms following chorea. The connection in this case between chorea and epilepsy seems close, while the duration of the chorea, its rather abrupt termination, with the almost immediate development of epilepsy, together with the absence of any constitutional vice, makes the connection more probable.

In conclusion, my thanks are due to Dr. H. C. Harris for the privilege of reporting these cases, and also to Dr. Eliot Gorton and Dr. L. L. Mial for reference to cases in their wards.

Reviews.

ELECTRICITY IN THE DISEASES OF WOMEN, WITH SPECIAL REFERENCE TO THE APPLICATION OF STRONG CURRENTS.

By G. Betton Massey, M.D. Physician to the Nervous Department of Howard Hospital, etc., Philadelphia. F. A. Davis, 1889, pp. viii.—210 (price, \$1.50).

A book upon a subject which has attracted so much attention as the electrical treatment of diseases of women has, must prove of great practical interest and utility, inasmuch as a thorough summary and critical digest of our knowledge upon this subject was, prior to the appearance of this book, still lacking.

In the introductory chapter the author says, that the keynote to the progress in the gynæcological use of electricity has been the use of a single pole for treatment. That a full test of the practical utility of electricity in gynæcology, shows that it is an agent capable of being properly applied without the need of a great amount of technical skill and that the necessary skill can be readily gained by anyone, even the busy general practitioner. The main object of this book is to verify these statements. Certainly the author is sufficiently modest in his purpose, which he fully succeeds in accomplishing. His book is exceedingly practical, with an almost complete absence of theory. It is, however, an open question whether the theoretically well informed electro-therapeutist does not possess advantages which the practically educated one must forego.

Chapter II describes the apparatus required in gynæcological applications of the galvanic current. The graphite rheostat described as the "author's rheostat" should be called Rudisch's rheostat modified by Massey. A paragraph devoted to the use of the incandescent light current in medical work, is of decided utility.

Chapter III on experiments illustrating the physical qualities of galvanic currents, contains a number of experiments which are of a simple and practical nature and are, undoubtedly of great value to the beginner in the study of electricity.

Chapter IV treats of the "action of concentrated milliampère currents on organized tissues." Precisely what is meant by a milliampère current is difficult to understand. The term is not a good one.

The other chapters on the methods of treatment hardly call for a review in this Journal. The book is one which is deserving of great praise and will prove of undoubted utility to every worker in this branch.

G. W. J.

Society Reports.

AMERICAN NEUROLOGICAL ASSOCIATION.

*Fifteenth Annual Meeting, held at Long Branch, N. J.,
June 26 and 27, 1889.*

DR. E. C. SEGUIN, President, in the Chair.

The following Scientific communications were read and discussed.

DR. GEORGE W. JACOBY, of New York, read a paper, entitled:

A CONTRIBUTION TO THE STUDY OF ANÆSTHETIC LEPROSY, WITH SPECIAL
REFERENCE TO PARTIAL SENSORY DISORDERS.

(For complete paper see page 336.)

The points to which particular attention should be paid in all cases were the electrical excitability of the muscles, the condition of the reflexes, the presence or absence of fibrillary twitchings, and the condition of sensation. Upon these data depended the diagnosis between anæsthetic leprosy and syringomyelia, as well as that of the central or peripheral localization of the leprous process. The conclusions arrived at by the author from the analysis of his own and other cases were that the differential diagnosis between the two diseases could not always be made; that partial sensory disorders are not characteristic of syringomyelia, but may occur in anæsthetic leprosy as well as in purely peripheral affections; and finally, that a differential diagnostic point between central and peripheral loss of temperature sense may lie in its complete loss in one case and its partial loss in the other.

DR. STARR said that the same question as to the differential diagnosis between syringomyelia and anæsthetic leprosy had only recently occurred to him. Three weeks ago he had seen a case with Dr. Prince A. Morrow, of New York, in which there had been a gradual onset of an atrophic and anæsthetic affection of the right arm. He came from the Sandwich Islands and has been exposed to leprosy. There was anæsthesia to temperature and pain, but not wholly to touch, along the hand and part of the arm. There were small reddish-brown spots on the arm, which Dr. Morrow considered

leprous in character. The idea of syringomyelia at once occurred to him, but the history of exposure in a leprous country sufficed to make anæsthetic leprosy at least the more probable nature of the disease; but without such history it would have been almost impossible to make the diagnosis. As to the matter of partial loss of sensation in the cases of multiple neuritis and beriberi he had seen, the sensory loss was complete, with the exception, however, of muscular sense.

THE PRESIDENT remarked that he had now under observation a case of leprosy, but had as yet made no careful examination of the temperature sense. His case illustrated the ease of diagnosis at an early period, when confusion with syringomyelia could not occur. The patient had a wine-colored eruption on the entire left leg and part of the thigh, the foot being free, and there were three or four similar spots on other parts of the body. There was distinct analgesia and some anæsthesia in these areas, but there was no paralysis or atrophy. The patient had come from the Sandwich Islands. The well-defined limitation of anæsthetic areas showed that it could not be neuritis. Of late the anterior leg muscles had become paretic, but there was no reaction of degeneration. The left hand was beginning also to manifest similar symptoms. There were no ulcers.

DR. C. K. MILLS had seen two cases of leprosy. He thought the author's point with regard to partial sensory disorder very interesting, but he did not see why there should not be partial disturbance also in neuritis, and, in fact, in traumatic neuritis it was quite frequent. The nature of the sensory disturbance depended upon the extent of the injury to the nerve. In ordinary multiple neuritis it might be true, as Dr. Starr insisted, that there was complete loss of sensation, through destruction of all the sensory fibres. He did not see, however, why the sensation to cold should not be injured as much as any. It should be borne in mind that in all infectious diseases attacking the nervous system there was a tendency to seize upon the central as well as the peripheral portions at the same time.

DR. GRAY agreed with the last speaker as to partial sensory disorders met with at times in peripheral neuritis. He had observed them also in multiple neuritis, where, for instance, there was impairment of the touch and temperature senses, yet the pain was excruciating. He did not see that it was easy to diagnosticate syringomyelia, and Wichmann and Starr had given no rules for diagnosis in their pamphlets. He believed that no one had made a diagnosis of syringomyelia in life.

DR. STARR stated that Schulze had made the diagnosis in three cases, which had been substantiated by autopsy. The points for diagnosis were the general conclusions drawn from a study of collected cases. Anna Bäumlér had brought together one hundred and sixteen cases, to which thirty or more had since been added.

DR. GRAY thought these diagnostic points would apply to many other spinal lesions.

DR. BIRDSALL had had ideas similar to Dr. Gray's as regarded the diagnostic indications in syringomyelia until he had read Roth's collection of cases. In a certain number of such cases there were clinical pictures differing altogether from those of other spinal lesions and from that of peripheral neuritis. Disturbance of the temperature sense was the particular characteristic. Still, the testing of this sense had been constantly neglected in studying other spinal cases, and it was possible that it might often be disturbed in other spinal disorders. It was hard to see how any other lesion could produce precisely the same symptoms as those of syringomyelia. It was a question whether tracts for temperature and pain could be localized in the cord. Peripheral nerve lesions might cause injury to some sense fibres and not to others, although generally all were injured, particularly where the inflammation was truncal in character. There might be partial sensory disturbance in dermal forms.

DR. GRAY said it was easy to localize disease in the anterior cornua, in the lateral and posterior columns of the cord, but the diagnosis of central lesions was very difficult. The fact that out of one hundred and fifty cases of syringomyelia only three have been diagnosticated during life proved the truth of his assertion. The diagnostic suggestions given were therefore empirical. The presence or absence of the temperature sense has not been sufficiently tested as yet. How would one distinguish a chronic central myelitis?

DR. BIRDSALL stated that central myelitis began acutely, and thus differed from the slow advance of syringomyelia.

DR. MILLS thought the question of partial sensory disorder most interesting. But he thought that there would be more apt to be partial disturbance in a truncal than in a dermal neuritis.

DR. FREDERICK PETERSON, of New York, asked the president if the tendon reflexes had been exaggerated in his case of leprosy, and was answered in the affirmative. He had asked this because he had recently read an interesting study of the reflexes in anæsthetic leprosy made by Dr. Suzuki, of Tokio, and published in the "Sei-a-Kwai" medical journal, in which there was an analysis of

seventeen cases. One patient had normal reflexes, while all the others had increased tendon jerks, and in some of the cases there was even ankle clonus. The conclusions arrived at by the author were similar to Dr. Jacoby's.

The PRESIDENT called attention to the fact that so many cases of leprosy were now being continually imported into the United States. It seemed as if the country was threatened with its domiciliation. It was a question whether it was not the duty of physicians having such cases to report them to the authorities.

DR. WHARTON SINKLER, of Philadelphia, mentioned the recent presence of two cases in Philadelphia. The physician in charge had been fined by the Board of Health for not reporting them.

DR. PRINCE wondered at the manner in which leprosy patients were allowed to go about, and thought also that the attention of the authorities ought to be called to the disease, and reports to the boards of health be required.

DR. GRAY knew that in the East, where the disease was prevalent, isolation was practiced. He wished to know what the actual danger of contagion was.

DR. BRILL said that the attention of the New York Board of Health had already been called to the matter.

DR. JACOBY said that Schulze was altogether too positive in his assertions. This author went so far as to contend that cases of leprosy, where cavities had been found in the cord, were not leprosy at all, but syringomyelia. As for himself he thought a central lesion probably often existed in this disease, although it was well-known that the essential pathology lay in a truncal neuritis. A simple dermal neuritis could not be assumed in leprosy. In his case, for instance, the indurated and swollen ulnar nerve could be distinctly felt. He did not agree with Dr. Gray that the diagnosis of syringomyelia could not be made. The only danger lay in mistaking it for leprosy. He had only last evening come across the work of Suzuki mentioned by Dr. Peterson, but had noticed the identity of that author's conclusions with his own, although they had been arrived at from different points of view.

DR. J. H. LLOYD, of Philadelphia, read a paper entitled :
A LATER HISTORY OF A CASE OF FOCAL EPILEPSY, FOR WHICH TREPHIN-
ING AND EXCISION OF THE MOTOR CENTRES WERE PERFORMED.

(For complete paper see page 356.)

Full details of the case had been recorded at the last annual meeting of the Association. At that time but three months had

elapsed since the operation, and Dr. Ferrier, of London, had stated in a discussion that the time was too short for a decisive result as to the usefulness of the operation. Now that more than a year had passed, further details could be given. Before the operation the patient had had many seizures daily. There was no fit for four months after the operation. Since that time, however, there had been some ten seizures in nine months.

DR. SPITZKA said there was a minority, but a powerful minority, of clinicians and physiologists who hesitated to accept the dictum that spasms were always of cortical origin when clonic in character and local in manifestation. There were well-authenticated cases of spasms, such as are generally described as cortical, which undoubtedly had their origin in the pons or medulla. He therefore thought it premature to advise surgical procedure when there was still doubt as to the position of the convulsive centre. He was of opinion that the seat of irritation in the great majority of cases of epilepsy was in lower centres, such as Nothnagel's convulsive centre.

DR. MILLS mentioned the case of a man subject to unilateral convulsions beginning in the hand. There was a scar upon the head, and trephining was done at this spot in order to relieve severe pain, probably due to trigeminal irritation in the scalp or dura. This operation relieved the pain, but caused the Jacksonian epilepsy upon the other side. The operation was repeated in the same spot, the membranes and cortex being removed to some extent, with great success. It was an apparent illustration of the fact that convulsions may occasionally be the result of operations themselves.

DR. DERCUM had seen the same case, and stated that the dura was not opened in the first operation. At the second trephination the membranes were found united. There was a pachymeningitis. The patient had surgical epilepsy. Six months elapsed between the operations.

DR. LLOYD said it was a question whether one could have localized epilepsy from irritation of lower centres, as Dr. Spitzka had intimated. He had always held the idea that such spasms were of cortical origin. In the case he had just described the absence of fits for four months led him to believe that he had removed the parts concerned in their manifestations. He had been hopeful of the efficacy of this method in the relief of such a disorder, and was not yet willing to give up the idea that something may be accomplished in this way. He thought it would be justifiable to operate again in this same case.

DR. SPITZKA asked if the cortex removed had been examined microscopically, and what was the pathological condition found.

DR. LLOYD answered that the microscopist had reported atrophy of the cortical cells.

DR. SPITZKA remarked that atrophied cells could scarcely be very potent in the production of epilepsy.

MULTIPLE NEURITIS AND INFECTIOUS CEREBRO-SPINAL MENINGITIS.

(For complete paper see page 351.)

DR. H. S. UPSON, of Cleveland, read a paper in which he considered the relation between these affections. Dr. Mills had first suggested the possibility of a connection between the two. His own case was briefly as follows: A woman, aged twenty-seven, had intense pain and tenderness in the extremities, together with stupor and slight opisthotonos. No electrical examination could be made, because of the extreme hyperæsthesia. She had had a multiple neuritis a year before, but recovered. The autopsy revealed congestion of the pia of the brain and cord, with marked serious effusion, and a microscopic examination of the ulnar nerve revealed interstitial inflammation there. In the opinion of the author, the nerve trunk inflammation was not parenchymatous, but rather of the membranes, and was analogous to the process in the brain and cord; the nerve fibres were involved secondarily. He had not looked for bacteria.

DR. MILLS naturally felt interested in the case. He had had an autopsy in one of his own cases. Portions of nerves and sections of the brain and cord had been examined by Dr. Gray, of Washington, and this examination had proved conclusively the association of neuritis with the cerebro-spinal meningitis.

DR. SPITZKA referred to the vacuoles in the author's sections, and said retarded lymph outflow might be important in causing the death of tissue and the formation of gas bubbles which these vacuoles must be considered to represent. He had noted also two bodies in the section larger than ordinary leucocytes, very delicately stained, which the author should have carefully delineated.

CASE OF ABSCESS OF THE BRAIN; OPERATION; DEATH ON THE NINTH DAY.

(For complete paper see page 329.)

DR. C. K. MILLS, of Philadelphia, gave an abstract of a paper on this subject by Dr. J. T. ESKRIDGE, of Denver. The patient had had typhoid fever. Two months later there was purulent inflammation

of the middle ear, and not long after symptoms of brain irritation followed, such as headache, delirium, and persecutory delusions, together with paralysis of the right hand and the right angle of the mouth. The skull was trephined over the face and hand centres, and a purulent inflammation was found under the dura. The wound was then dressed, and the patient died nine days later. A widespread suppurative meningitis was found at the autopsy. Dr. Mills thought the case of great practical value. He thought trephining should have been performed in two places—at the center, which had been properly localized, and also over the mastoid region, where the inflammatory process had originated.

PERISCOPE.

BY DR. N. E. BRILL.

PHYSIOLOGY OF THE NERVOUS SYSTEM.

A CONTRIBUTION TO THE STUDY OF THE PERIODICAL PSYCHOSES. By E. Mendel, (Berlin) Allgemeine Zeitschrift f. Psychiatrie, etc. Bd. xliv., heft. 6.

After giving a history of the description of this form of alienation beginning with Aristotle and terminating with von Krafft-Ebbing, the author adds in addition to the following forms, a fourth group which he calls Periodic Paranoia. These forms are as follows :

1. Periodical mania.
2. Periodical melancholia.
3. Periodical Hallucinatory Delirium.
4. *Periodical Paranoia.*

The accompanying histories will serve to distinguish this last named group.

Case 1. H—, 33 years old, merchant. No obtainable proof of hereditary taint ; was always well until five years ago. At that time, after business annoyances and cares, the first attack of psychological disturbance appeared, which regularly returned at intervals of four months.

The attack begins usually, without a defined premonition, with insomnia and a rush of blood to the head, with red face and congested eyes, darting pains in the temples, etc. Then he hears voices in the street and in his room calling his name, and speaking about him ; he has a sweet taste in the mouth (in one attack 0.5 per cent of sugar was found in the urine); refused food for a few days, and had the delusion that his relatives and wife were conspiring to poison him. He kept a revolver at nights under his pillow to defend himself against his enemies. Notwithstanding he attended to his business, he related to his customers that he was the ablest merchant, that he saw a good deal of the world, and that his competitors were stupid. At the Emperor's jubilee he sent to

him (he was a small merchant in a small town) a congratulatory message written on white satin, and said that he would be decorated for it. In one of his attacks he said his father was the son of Ballachini, and that thereby he himself was in possession of all the latter's secrets.

No motor disturbances were observable. If he were not removed he would attend to his business during his attacks. In addition to his delusions he was distrustful, depressed, and at times emotionally exalted.

The attack itself lasted three to four weeks, gradually disappearing, leaving the patient moody and distrustful. Then he retains a full knowledge of his illness, which he describes as a persecuting insanity.

Case 2. Miss Z—, 25 years old. No history of insanity in the family, although the mother is an easily excitable, nervous woman. Since six years, at pretty regular intervals of a year, attacks appeared, the last of which the author saw. This one was said to be identical with the previous ones.

The attack begins with visual, auditory and olfactory hallucinations. The patient draws away from her former surroundings, speaks very little, at times refuses food, is usually quiet and keeps to her bed. The delusions, which can only be brought out with great difficulty, are as follows :

She believes that she is persecuted by the Emperor, who wishes to arrest her, and she saw on the street how the gendarmes followed her, therefore she does not dare to go out. The Emperor does this in order to prevent her taking the rank to which she was born, viz. : a princess, who had been put under the charge of a mother who was not her real mother. The attacks last three to four months, and usually end quite suddenly. After a short time every trace of the attack vanishes.

Case 3. A woman, 51 years old, widow, with an aunt on the mother's side insane, and a son of the same aunt likewise. She was always healthy up to the time of first attack, and has three children who are healthy.

The delusional history is almost identical to that of case 2, viz. : born in high rank, brought up by a woman who, although asserted to be her mother is not her mother ; one of her own sons is the crown prince, and she is the daughter of the Emperor, who repeatedly visits her in her room. Her children were exchanged

through an opening behind a mirror in the wall of her room, etc. She loses flesh during her attacks and frequently refuses food. However, she is very quiet, and in one attack, treated at home, she suddenly became well. She explained one morning that during the night she became suddenly better, and that everything which she said had been nonsense. The attacks ending in this manner re-appeared four times in as many years.

These cases showing a combination of persecutory and grandioso delusions in the absence of somatic changes, without epileptic or hysterical antecedents or concomitants, are unknown in mania as in melancholia, and are characteristic of paranoia. That there cannot be relapses of the same condition the author asserts for the same reasons which he adduces in differentiating a relapse in mania from a periodical mania. (Die Manie, p. 75.)

THE INCREASE OF PARALYTIC DEMENTIA. By Dr. Otto Snell (Munich). *Allgemeine Zeitschrift f. Psychiatrie*, etc. Vol. xliv., bd. 3 ; heft. 6.

It is very doubtful whether the oft-expressed assertion that this century has been characterized by an increase in mental diseases, and by those conditions to which the names of neurasthenia and nervousness have been applied, is true. The fact that hospitals and asylums show an increased number of patients is no proof, for such was the condition of asylums and the treatment of its inmates that people rightly refrained from sending any of their relatives to such places. With the improvement in the care of the insane, confidence in asylums increased so that finally their advantages were called into requisition, and patients who were formerly kept at home and did not come under the observation of alienists were soon transported to those institutions. Under these conditions a much larger number of the insane came under treatment than formerly, and since statistics of the insane treated outside of asylums give but uncertain results, it becomes very difficult to determine what proportion of this apparently rapid increase in psychical disturbances is to be ascribed to these different conditions.

But of the increase in progressive paresis the author thinks there can be no doubt. This view is taken by Lanier, Hask, Tuke. Reinhard comes to a similar conclusion by comparing the statistics at Hamburg, where, between the years 1871 and 1883, the number of insane in comparison to the number of sane only increased to the smallest extent, the number suffering from paralytic dementia in comparison with all the other insane, was greatly increased, so that in the year 1877, there was one paretic to 7.5 non-paretic, while in 1883 this relation was as 1 to 4.

Opposed to these results are those of Clouston, who denies the increase in Edinburgh, and asserts that there was a decrease in paretics from 7.3 per cent, which was the percentage suffering from that malady in 1877, to 4.5 per cent. in 1885. Hurd and Sommer corroborate him.

The author compares the number of admissions of paretics with the number of admissions of all other insane in single years, then averages the total. He gives a table with yearly statistics for the last thirty years, the summary of which is as follows :

Admitted—Men, 2469, of which 383 were paretics ; hence 15.5 per cent.

Women, 1894, of which 47 were paretics ; hence 2.5 per cent.

Total number, 4363 ; 430 paralytics, or 9.9 per cent.

From this he concludes that there was almost double as many paretics during the past thirty years than in the preceding thirty.

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SEE "MERCK'S INDEX," PAGES 106 AND 167

THE
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Nervous and Mental Disease.

Original Articles.

A REPORT OF A CASE OF SYRINGO-MYELIA.¹

By IRA VAN GIESON, M.D.,

First Assistant at the Laboratory of the Alumni Association of the College of Physicians and Surgeons, New York.

FOR the opportunity of presenting this case I am indebted to Professor Francis Delafield, who gave me the material, with the clinical history and his notes of the autopsy.

Clinical History.—Lawrence B——; farmer; æt. 47; parents died of consumption; had been in good health with the exception of one slight attack of acute rheumatism in the hands until twenty-one months before death. He then noticed a soreness in the epigastrium and a numbness in the left foot and leg. These symptoms grew worse, and the right leg and foot also became numb.

For nine months after the commencement of the symptoms, the man was able to work on a farm, but could not do heavy lifting, and the jar of riding in a wagon made him numb from his waist down. At the end of this time he began to have a girdle sensation, and the motor and sensory symptoms in the legs had increased until he was hardly able to walk alone. The sphincters were normal and there were no symptoms in the upper part of the body.

During the *twelfth month* of the illness the condition was as follows: Occasional temporary incontinence of

¹ One of the essays for which the Cartwright prize was awarded in 1889.

urine lasting a day or two. Sensation normal in the back, but diminished in both legs, especially the left. Patellar reflexes slightly exaggerated, plantar reflexes diminished. Diaphragmatic and cremasteric reflexes absent. Patient cannot walk alone and drags the left foot. Romberg's symptom present. Ankle clonus absent. No rigidity of the legs. There were also periods of temporary improvement during this month.

During the *thirteenth and fourteenth months* of the illness the following notes were recorded: Patient remains in bed most of the time. He can walk only with firm support; but on some days there is transient improvement, and he is then able to be up and move about. The sphincters are controlled, except occasionally. There is daily œdema of the feet, subsiding at night. The girdle sensation is diminished, and is at times absent. The patellar reflex is at times normal, at times exaggerated; the diaphragmatic reflex is sometimes present, sometimes absent; the plantar reflex is diminished, the cremasteric is absent and the ankle clonus is present.

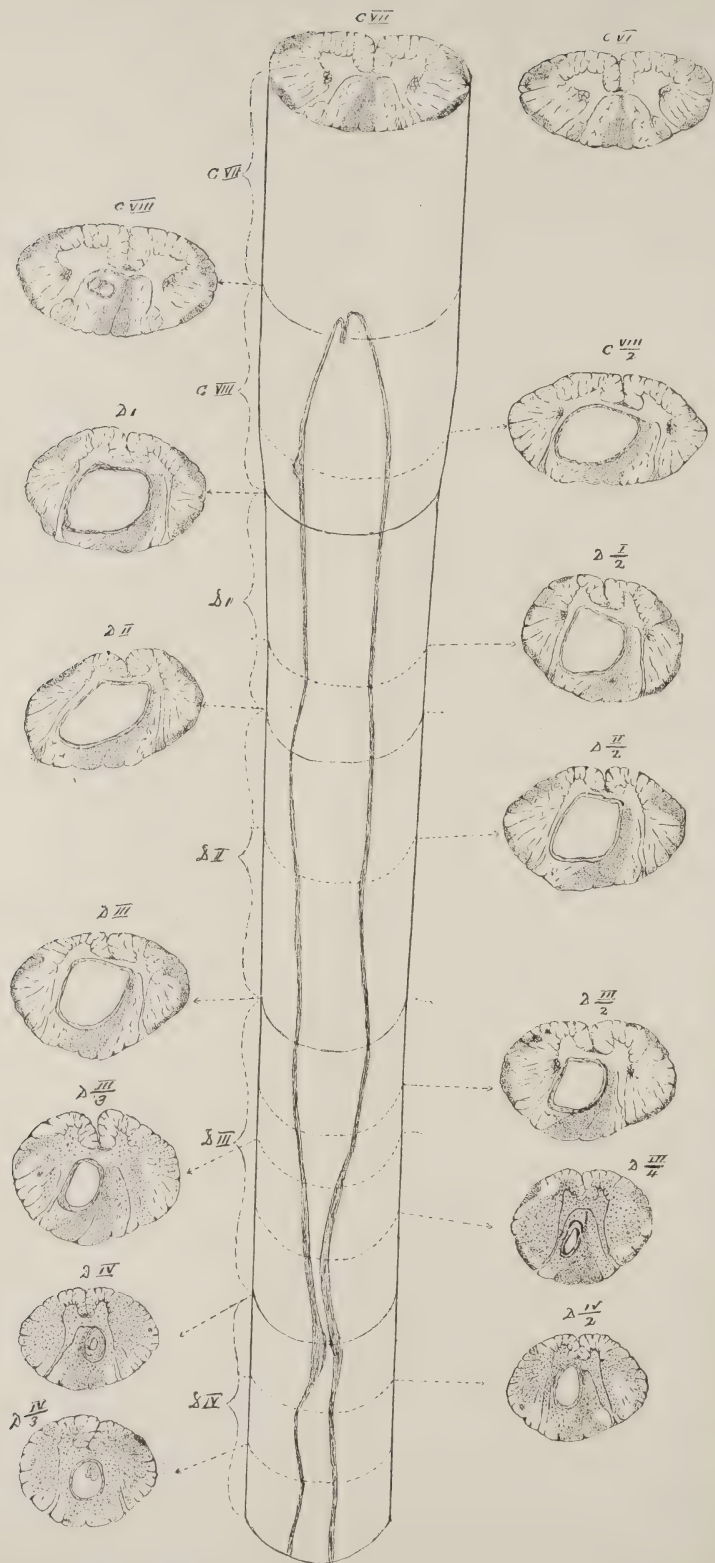
From the *fifteenth to the eighteenth months* of the disease, there was very little change in the man's condition. The daily œdema of the feet, subsiding at night, continued. He could stand for a short time with his feet far apart. Constipation and slight dribbling of urine. The girdle sensation was constantly present. There was exaggeration of the patellar reflexes, and diminution or absence of the cremasteric reflexes. The nutrition was good. Sensation above the umbilicus was normal.

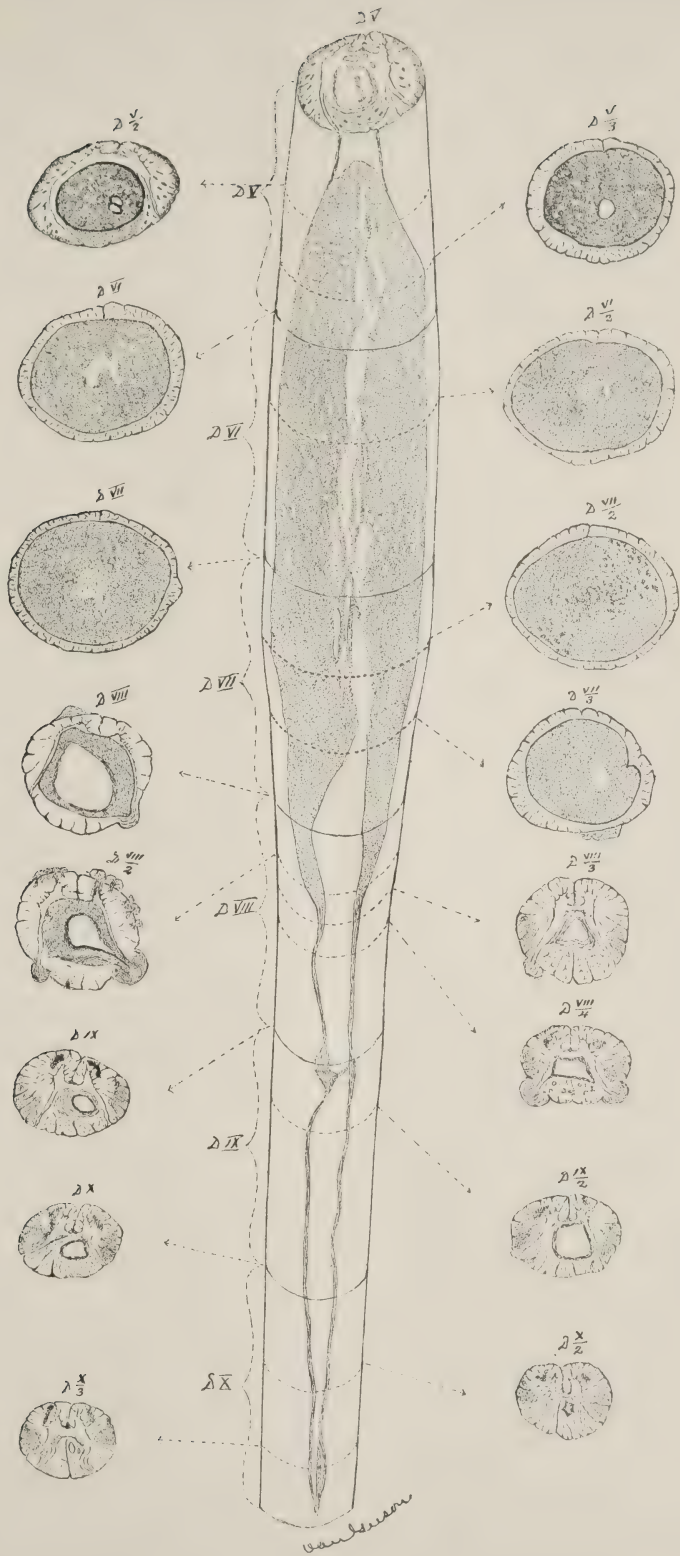
During the *nineteenth and twentieth months* there was in addition to the other symptoms considerable pain at times in the feet, and in the umbilical and hypogastric regions.

During the *last month* of the sickness bed sores developed, and the legs became very much swollen and œdematous. Four days before death the temperature rose to 103.5. The symptoms did not extend above the umbilicus.

Autopsy.—Body moderately emaciated. The thoracic and abdominal viscera were normal except the right lung, which was coated with fibrin, and the bronchi of its un-

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aerated lower lobe contained pus. A fusiform enlargement of the spinal cord, caused by a reddish gray tumor filling up a large part of its transverse diameter as a solid plug, begins near the middle point of the dorsal region, and extends downward four cm. Extending about eight cm. above the tumor and about five cm. below it, is a centrally situated tubular cavity with a thin smooth translucent wall, varying from two mm. to three-quarters of a centimetre in diameter.

Projecting from the surface of the cord over the lower half of the tumor are about sixty small grayish white nodules, from one to one and a half mm. in diameter. Some of them, the smaller ones, lie scattered about on the posterior and lateral surfaces of the cord, and are fused with the root strands. A few of them spring from the cord at the exit of the anterior nerve roots. The majority of the nodules, including the larger ones, surround the filaments of the posterior roots at or near their entrance into the cord. Several of the nodules in the latter position extend for some distance (about two segments) below the tumor. The brain could not be examined. The cord was hardened in Müller's fluid, and afterwards in alcohol; it was then divided into segments corresponding to the insertions of the anterior nerve roots. The segments were carefully measured, and Fig. 1 was constructed from these measurements.

Microscopical Examination.—Above the tumor (Fig. 1), the posterior median, the direct cerebellar column, and the antero-lateral ascending columns of Gowers are degenerated; below the tumor there is a degeneration of the crossed pyramidal tracts. Above the upper third of the fourth dorsal segment, the inner four-fifths of the column of Goll are deflected to the right by the large cavity. The column of Gowers is larger on the right side in the sections D II-2 and D III (Fig. 1). In the other sections these columns are nearly symmetrical and equal.

The upper limit of the cavity is about two mm. above the superior surface of the eighth cervical segment (Fig. 1). The uppermost portion of the cavity is lined by a thin mem-

brane of neuroglial tissue, composed of larger and smaller branching neuroglia cells and small spheroidal and oval cells lying in a network of fibres which unites with the surrounding neuroglia with a more or less distinct line of demarcation (Fig. 2). In some places the elements of this membrane are packed closely together, so that in section it has a rather dense appearance; but in the greater portion of its extent the cells and fibres are loosely arranged, so that it has a porous meshwork structure as in Fig. 2. This



Fig. II.

thin membrane—which, for the sake of brevity, will be spoken of as the limiting membrane of the cavity—is the simplest form of the wall of the cavity, and is the only wall the cavity has in a considerable portion of its extent, viz., in the upper two-thirds of C VIII., in D I., D II., in the upper third of D III., and in the greater part of D IX. and D X.

This limiting membrane also lines the tubular cavity in the lower third of the tumor.

In other places the wall of the cavity is in a state of hyperplasia and is more complex in structure. In these places (indicated by the thickened vertical outline of the cavity in Fig. 1) the limiting membrane is sometimes present and sometimes altogether absent.

In the section C VIII. (Fig. 1) the cavity is bifurcated, appearing in the apex of the column of Burdach as two small circular spaces (1 mm. in diameter) surrounded by

the limiting membrane. The gray matter with its ganglion cells is normal, except that a large inner portion of the cervix and head of the posterior horns is replaced by a closely tangled, very fine filamentous network containing larger spider cells and spindle shaped cells with long, brightly shining bipolar processes. This is most prominent in the left caput cavu posterior. With carmine these gliomatous areas in the gray matter stain more deeply than the surrounding gray matter.

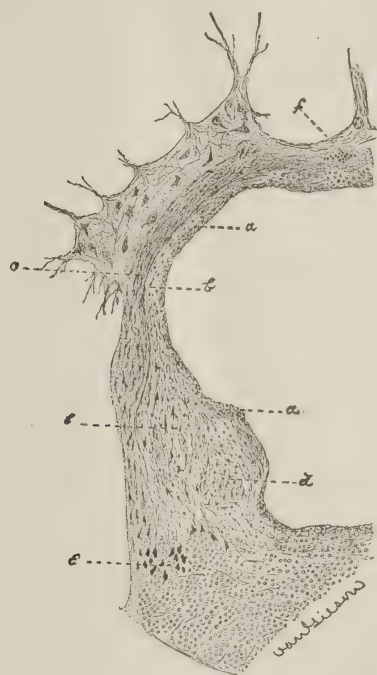


Fig. III.

In the section C VIII.-2 (Fig. 1) the cavity (6 mm. in diameter) is lined with the limiting membrane, and is bounded by the posterior commissure, the deflected column of Goll, and the left column of Burdach. The left hand portion of the wall of the cavity is in a condition of hyperplasia (Fig. 3), which makes at one point a slight nodular projection into the lumen.

On the left side of the cavity the limiting membrane merges into a zone (Fig. 3) of delicate, approximately parallel filaments containing a few larger and smaller branching neuroglia cells, and many small round and oval cells which are so intimately associated with the filaments that it is difficult to determine whether they have processes. Some of these small round cells are very likely, in such a growing mass of neuroglia, immature forms of the larger branching neuroglia cells.

The posterior portion of this filamentous zone envelops a little ball of small branching neuroglia cells whose fine processes form a loose network. This zone replaces entirely the inner four-fifths of the left column of Burdach,

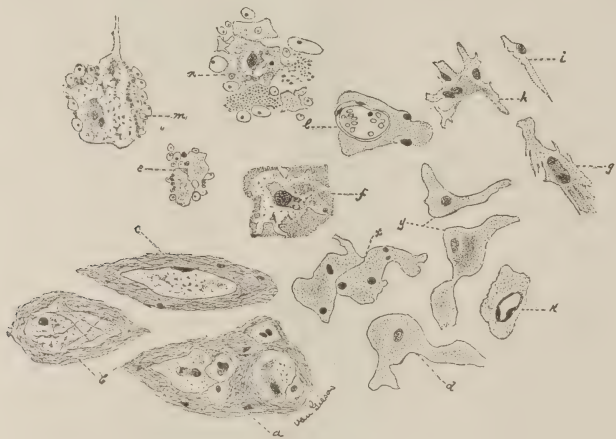


Fig. IV.

and involves to the extent shown in the drawing (Fig. 3) the gray matter where there are more neuroglia cells—most of them spindle-shaped with long bipolar processes—and fewer filaments. The right posterior horn is similarly but less extensively involved.

The hyperplastic portion of the wall also contains some small holes and spheroidal cavities, which apparently are the result of a liquefactive degeneration, and disappearance of the neuroglia cells. This seems to be so, because the various stages of the replacement of the cell-bodies by fluid are present. In some of the cells the nucleus is attached

to or surrounded by the homogeneous remains of the cell-body, and what is left of the cell-body is surrounded by fluid which stains poorly with acid fuchsin and carmine (Fig. 4 a). Other cells are converted into cavities filled with fluid (Fig. 4 b c). The gliomatous portion of the gray matter and the degenerated column of Goll contain glia cells liquefying in this way and producing little cavities. (I do not think these changes in the glia cells are produced by the action of the hardening agents, because the degenerating cells are not scattered about uniformly throughout the cord but are noticeable wherever there is much *hyperplasia* of neuroglial tissue, and in the œdematous portions of the cord described presently.)

In the apex of the left posterior horn and in the adjacent portion of the column of Burdach (Fig. 3) is a group of very much swollen and partially fluidified large glia cells, and some large irregular shaped drops of fluid. The swollen cells (Fig. 4 d) are changed into homogeneous rounded or irregular shaped nucleated lumps, occasionally having a thick, varicose, club-shaped process. Others of the degenerated cells are more or less completely changed into fluid. It is difficult to determine whether some of the large irregular drops of fluid are the result of complete liquefaction of the Deiter's cells, or whether they have perhaps leaked out of the blood-vessels. One of the smaller vessels in this region has a partial zone of fluid about its walls.

Between the sections C VIII.-2 and D III.-3 (Fig. 1) the wall of the cavity has the simpler form shown in Fig. 2. The posterior horns and the internal portion of their junction with the anterior horns, in this portion of the cord, have an increased amount of neuroglia cells and fibres. This is most marked on the left side.

In the section D III.-2 the limiting membrane is replaced by a considerably thicker zone, resembling the superficial neuroglial layer of the cord, wrapped circularly about the cavity and merging with the surrounding thickened neuroglia of the posterior columns and gray matter.

The section D III.-3 is near the upper level of a portion of the cord, in which the central portion is very œdematous.

The œdema extends nearly to the fifth dorsal segment. The wall of the cavity in this section is thickened and has about the same structure as in the preceding section. It contains liquefying glia cells and some drops of fluid. The gray matter and the adjacent portions of the lateral columns and the inner half of the posterior columns are honeycombed with drops of fluid and some granular material, which separate the elements of the gray matter and crowd apart the nerve fibres in the white matter. Besides this there are a great many swollen and liquefying glia cells (Fig. 4 y) in the œdematous areas and in the wall of the cavity. These cells are changed in the same way as already described in the section C VIII.-2. Many of them are so completely liquefied that they are not to be distinguished from the drops of fluid which have exuded from the blood-vessels. The latter are congested, and in a few places, notably in the gray matter, have several leucocytes with trifoliate nuclei in their perivascular spaces. Some of the ganglion cells are partially disintegrated.

The section D III.-4 the wall of the cavity is thickened and is composed of two zones, an inner sparingly nucleated filamentous zone wound about the cavity circularly, and an outer incompletely circular layer composed of long-armed, spindled-shaped neuroglia cells lying amongst small bundles of rather stout, glistening fibres which have a vertical direction.

In the section this outer zone has a peculiar appearance and is different from any other portion of the wall of the cavity, because the fibres are cut transversely and appear as collections of larger and smaller homogeneous, lustrous, angular discs (from 1-8 μ in diameter) separating the spindle cells. Attached to the inner wall of the cavity are some red blood-cells and a few cells looking like leucocytes. The œdema and the liquefactive condition of the glia cells is more intense and extensive than in the preceding section.

The degenerating glia cells are most prominent in the posterior horns, where in some places the swollen cells are nearly in contact (Fig. 4 x) and in other places some of the

cells seem to have coalesced, forming large, irregular shaped, faintly stained, homogeneous, nucleated lumps.

Lying against and around a very few of the smaller blood-vessels in the œdematous areas, are some homogeneous lumps of material (Fig. 4 k, l) having the same appearance as the swollen glia cells. These perivascular homogeneous lumps probably are swollen glia cells which happened to lie very close to the blood-vessels.

In the section D IV.-1 the gliomatous wall of the cavity

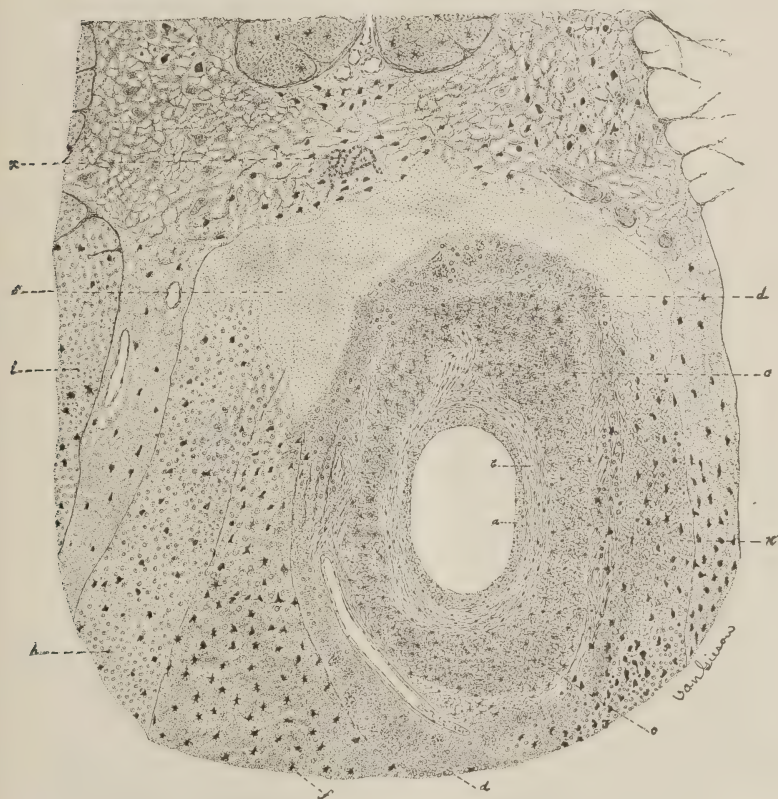


Fig. V.

containing numerous new thin-walled blood-vessels and fluidifying glia cells (Fig. 4 g, h, i) is in a more pronounced state of hyperplasia than in any other portion of the cord. The details of the structure of the wall are given in the text

of Fig. 5. About 1 mm. below this section the wall has grown into the cavity, so that the lumen is of microscopic size: The œdema and the changed glia cells are present as in the previous section. The œdema is greatest in the gray matter adjoining the commissure.

Between the outer portion of the wall and the gray commissure, and replacing a portion of the latter, is a crescentic zone consisting largely of fluid (Fig. 5), which seems to have collected from the gray matter about the dense and comparatively impervious outer layer of the cavity wall partially separating it from the surrounding tissue. This mass of fluid disappears at a slightly lower level. The wall of the cavity contains a little fluid in its meshes.

The remainder of the sections from the fourth dorsal segment do not differ materially from the previous section (D IV-1), except that the cavity gradually grows larger and its wall is much less thickened and has a simpler structure. It is composed of fine filaments with few cells, wrapped concentrically about the cavity, and the filaments merge into the surrounding thickened neuroglia. Clinging to the inner surface of the wall in the lower part of this segment (Fig. 1 D IV-4) is a mass of fluid and fibrin. The œdema ceases at the upper level of the fifth dorsal segment. The œdema seems to be recent in its origin, because it has only separated and not destroyed the elements of the cord to any great extent except the glia cells. The posterior horns and the gray commissure in the œdematous region were probably in a condition similar to that shown in Fig. 3 before the œdema took place. This is indicated by the great numbers of the changed glia cells in these œdematous portions of the gray matter (Fig. 5).

The section D V-1 shows the lesions of a chronic myelitis of the white and gray matter. The wall of the cavity, containing the changed glia cells, has about the same structure as in the lower portion of the fourth dorsal segment. A strand of fibrin stretches across the cavity.

Three mm. below this section is the upper dome-shaped extremity of the tumor (Fig. 1), which gradually growing larger and compressing the tissue of the cord into a thin

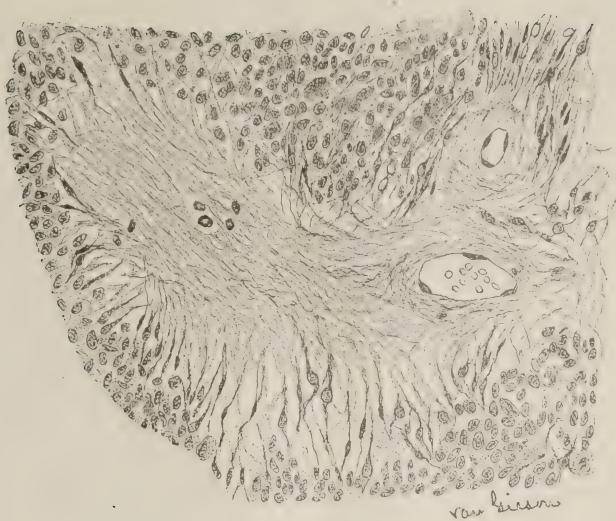


Fig. VI.

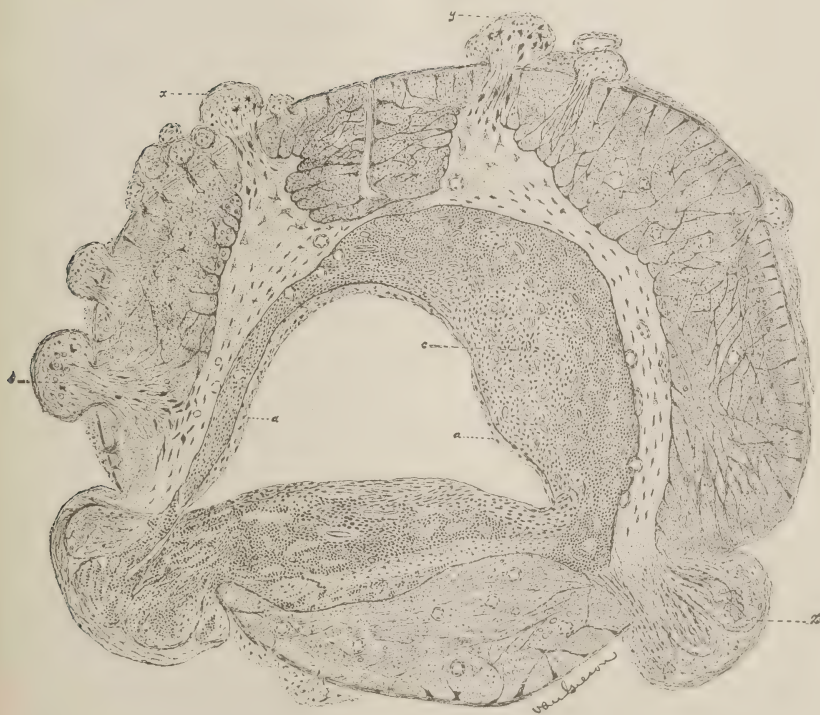


Fig. VII.

cylindrical outer shell attains its maximum diameter at D VII, where the cord measures sixteen mm., nearly twice the usual diameter of this transection.

In section, the tumor is composed of closely packed masses of spindle-shaped and oval cells associated with filaments, arranged in strands between and around numerous blood-vessels which are surrounded by a close-textured sparingly nucleated sheath of neuroglia filaments (Figs. 6, 7). When isolated, some of the tumor cells are glia cells, and other granular, rounded, or spindle-shaped non-branching cells, look like the cells of the ordinary sarcomata. The central core of the tumor is degenerated. It is composed of fluid, fibrin, hæmatogenous pigment, and, in places, of disintegrated tumor cells.

In the eighth dorsal segment the tumor does not fill up the large central portion of the cord as a solid plug, but is situated around a tubular cavity (Figs. 1, 7). Here the tumor is very distinctly separated from the cavity by a thin neuroglial membrane bearing no structural resemblance to the tumor, but identical in structure with the limiting membrane (Fig. 2). The tumor thus arranged about the cavity becomes thinner and thinner (Fig. 1), and about seven mm. below the section D VIII-2 disappears, fusing with the gliomatous wall of the cavity. The gray matter in the eighth dorsal segment, contains an increased amount of neuroglia, and there are patches of sclerosis in the white matter.

The small nodules on the surface of the cord are of three kinds (Figs. 1, 7).

In one place such a superficial nodule is produced by the stretching of the superficial neuroglial layer and a pouching out of the substance of the cord in front of a mass of tumor cells advancing toward the periphery (Fig. 1 D VIII).

Several of them are caused by direct nodular extensions of the tumor (Fig. 6).

The remainder are caused by little tumor-like hyperplasias of the cells and fibres of the neuroglia of the white matter. The latter are collected together in dense bundles

of various sizes near the periphery of the cord, and piercing the superficial neuroglial layer and pia mater covering of the cord, both the inner and outer extremities of these bundles spread out like the fibres of a broom (Fig. 7). The outer ends become tangled up in the pia mater producing little nodules, or they become fused with the neuroglia of the nerve roots which is increased at such points, and is composed of interlacing skeins of filaments with few glia cells, surrounding the nerve fibres which are diminished greatly in number. The inner ends of the bundles spread out in the gray and white matter becoming continuous with surrounding neuroglia. Between the separated ends of the fibres are generally a number of larger and smaller neuroglia cells, some of which are liquefying and have produced small cavities empty or filled with fluid (Fig. 7 b). A hypertrophy of the neuroglia accompanying the intra medullary continuations of the nerve roots forms the greater number of this third variety of the nodules.

The prominences on the roots at some little distance from the cord are due to a growth of neuroglial tissue.

In the eighth dorsal segment below the tumor the wall of the cavity containing several large blood-vessels and many liquefying glia cells, consists internally of a thin porous loose-textured inner layer and externally of a sparingly nucleated filamentous dense circular zone, which is somewhat thickened throughout the whole segment. A slender band of neuroglia stretches partly across the cavity at one place.

There are some short fibres in this portion of the wall of the cavity which have the appearance of hyaline degeneration. They are irregularly swollen, glassy, and stain brightly with dilute solutions of acid fuchsin.

The posterior horns, and the columns of Clark in places, are changed into neuroglial tissue. There is a neuroglial thickening about the posterior septum, and there are patches of sclerosis about the blood-vessels. Two columns of interstitial myelitis lying alongside of the external antero lateral margin of the anterior horns (Fig. 1) extend to the twelfth dorsal segment.

In the remainder of its extent the wall of the cavity does not differ materially from the wall in the first dorsal segment. The wall is nodularly thickened in one place (see Fig. 1 in the tracing of the cavity in D IX), and a neuroglial septum stretches across the cavity in the upper portion of the tenth dorsal segment. The right column of Clark in places is involved by the cavity wall. The cavity terminates a few mm. above D XI.

The central canal in most of its extent is normal; in places its cells are increased in number. In such places some of the cells are arranged in string-like rows, and others are grouped in a vertical tubular manner, so that in the sections they look like the lining cells in a transection of a small duct of a gland. Many of the vessels throughout the cord have a limited number of small round cells just outside of their walls. The œdema just above the tumor seems to be due to a passive effusion from the vessels. A tumor expanding the cord so extensively as this one would be liable, by interfering with the circulation, to cause an œdema in its vicinity.

Anatomical Diagnosis.—Telangiectatic glio-sarcoma occluding the middle portion of a tubular cavity in the cord, lined with neuroglia tissue which in places is in a condition of hyperplasia.

Resumé and Conclusions.—1. *There is no morphological evidence that the tumor extended beyond its present confines.* About two hundred sections of the cord above and below the tumor were examined, and in none of them is there any tissue having the structure of the tumor.

2. *There is no clinical evidence of the extension of the tumor above its upper level.* Since the symptoms did not extend above the umbilical region, the upper limit of the lesion is situated between the fourth and seventh dorsal segments.² This corresponds approximately to the situation of the upper level of the tumor. But the cavity is certainly large

² Localization of the functions of the segments of the spinal cord, in Professor Starr's paper on Syringo Myelia. American Journal of Medical Sciences, May, 1888.

enough ($\frac{3}{4}$ cm. in diameter) in the eighth cervical and first dorsal segments, had it been produced by the disintegration of a tumor, to have occasioned symptoms in the hand and forearm.

3. *The lower third of the tumor is developed outside of the inner zone of the neuroglial wall of the cavity.* In the eighth dorsal segment the tumor encircles the cavity, from which it is separated by a thin neuroglial membrane, structurally differing from the tumor, which interruptedly clothes the wall of the entire cavity internally quite extensively. This arrangement of the tumor suggests that it originated in the wall of the cavity. Possibly the upper two-thirds of the tumor grew into and occluded the lumen.

These three facts—the absence of morphological or clinical evidence of the formation of the cavity, by the tumor, and the intra-mural situation of the lower third of the tumor which is but slightly degenerated—indicate that the cavity was formed previous to the development of the tumor from the cavity wall. The cavity probably is a congenital defect; but as there are in places neuroglial hyperplasias of its wall with evidences of degeneration, the cavity has probably also been modified to some extent by the growth and disintegration of gliomatous tissue along its wall. In the places where the hyperplasia of the wall is greatest the lumen of the cavity is the smallest, and where the cavity is largest its walls are the thinnest. To what extent the cavity is congenital in its origin, and to what extent it has been enlarged by the growth and degeneration of gliomatous tissue surrounding the cavity, is difficult to determine. On account of the accommodation of the structures of the cord to the configuration of the cavity, in the developing and adult stages, no symptoms occurred until twenty months before death, when the tumor began to grow from the cavity wall. Then the regional distribution and duration of the symptoms correspond very well with the position and structural character of the tumor.

Defective closures of the developing central canal and posterior septum, with residual epiblastic tissue in the wall of the defective closure, would explain how the cavity is

formed in many cases of syringo myelia. In a section of the spinal cord of the human embryo at the sixth week, the primary central canal is relatively very large; it is diamond shaped, and extends from the anterior to the posterior surface of the cord. During the later stages of development, this primary central canal becomes divided into two portions, an anterior and a posterior portion. The anterior portion ultimately forms the permanent central canal; the sides of the posterior division coalesce and form the posterior septum. With faulty closure of either of these divisions of the primary canal there is generally some surplus epiblastic tissue lining the wall of the defect. This embryonal tissue is liable at some later time to grow, forming neuroglial hyplasias or tumors. These growths have a great tendency to disintegrate and make the cavity, which may be very limited at first, much more extensive. It is in this way very likely that many cases of syringo myelia originate; the cavity commences in a defective closure of the divisions of the primary canal (or of the apertures through which the embryonal vessels penetrate into the cord), and acquires a greater extent by the growth and disintegration of embryonal tissue which persists along the wall of the defect. Errors in the closure of the divisions of the primary central canal as a starting-point for the cavity, explain the great frequency of the situation of the cavity, in cases of syringo-myelia, in the posterior columns or central part of the cord. In one place in this case, the cavity looks as though it was due to a defective closure of the anterior portion of the posterior septum (see Fig. 1-D VIII.-3 to D IX.-2). In these sections the anterior portion of the posterior septum is absent. Throughout the remainder of the cord the posterior septum presents no anomalies.

It is fully in accordance with Cohnheim's theory that tumors should be associated with these cavities, starting in a faulty development of the cord, and that furthermore they would be liable to arise from the wall of the cavity; for preferably in the wall of such a congenital defect we would expect the presence of residual epiblastic tissue, or of an included portion of some other inappropriate embryonic

layer, from either of which a tumor might arise. From the former condition, a neuroglioma or a neuroglial hyperplasia; in the later condition some heterologous tumor might originate.

It seems justifiable, in reviewing this case, to say that some cases of syringo-myelia are cases of congenital tubular defects in the cord, producing no symptoms until a gliomatous hyperplasia of their walls, or a tumor arising in the wall, becomes large enough to injure the cord.

The liquefactive degeneration of the glia cells in this case is interesting in connection with the breaking-down process characteristic of gliomatous tumors and certain gliomatous hyperplasias, which is so prominent a feature of these growths when they occur with syringo-myelia. The changed glia cells are so uniformly and extensively present in the hyperplastic portions of the wall as to suggest that the disintegration and disappearance of the gliomatous tissue in syringo-myelia may be, in a measure, due to such a liquefactive degeneration of the glia cells.

In the œdematous portions of the cord the changes in the glia cells are probably to a great extent secondary to the œdema; but in other portions the degeneration of the glia cells is quite independent of the œdema.

Such a condition as the œdema in this case tends to modify the configuration of the cavity. In one place the œdematous fluid has sequestered a portion of the wall of the cavity.

The literature of syringomyelia is given in Professor Starr's paper already referred to.

EXPLANATION OF THE PLATES.

Fig. 1.—Transections from different levels of the cord and a tracing of the tumor and the cavity on the posterior surface of the cord. (The segments of the cord and their subdivisions were measured by compass, after hardening and the tracings and transections are three times the natural size—the latter were drawn with the camera lucida. The transections from D III-4 to D IV-3 have not been represented proportionately large enough). In the sections between and including D III-3 and D IV-4, the dotted areas represent the distribution of the œdema. The thicker portions of the tracing of the wall of the cavity correspond to the places where the wall is in a condition of hyperplasia.

In front of the left anterior horn in the section D VIII and attached to the posterior surface of D VII-3 are extensions of the tumor producing nodules on the surface of the cord. Corresponding to the apex of the left posterior horn in the section D VIII a superficial nodule is formed by the tumor pressing out the substance of the cord. The nodules on the anterior surface of the section D VIII-2 are hyperplasias of the neuroglia of the white matter.

Fig. 2.—A section of the limiting membrane of the cavity (from the section C VIII, Fig. 1). The upper surface bounds the lumen of the cavity, the lower margin is the junction of the membrane with the surrounding white matter.

Fig. 3.—A section—a little below the section C VIII 2, Fig. 1—showing hyperplasia of the wall of the cavity. a. a., the inner or limiting membrane of the cavity; b. b., zone of filamentous neuroglia replacing the left column of Burdach, containing at d a small cluster of small spider cells; c., a zone of neuroglia cells and filaments involving the left posterior horn and inner one-half of the anterior horn; f., central canal; e., group of swollen and fluidifying glia cells.

Fig. 4.—Showing the liquefactive degeneration of the neuroglia cells from various places in the cord.

a. A group of degenerated glia cells from the hyperplastic portion (Fig. 3, b. b.) of the wall in the lower portion of the eighth cervical segment. In three of these cells a fragment of the protoplasm attached to the nucleus is surrounded by fluid. The degenerated cells are surrounded by closely woven fine neuroglia filaments; b, c, cells from the same region completely converted into fluid; d, swollen Deiter's cells from the same level of the cord (Fig. 3, e); f, g, h, i, degenerated glia cells from the hyperplastic wall of the cavity in the lower portion of the third and upper portion of the fourth dorsal segments. The remains of the cell-bodies are surrounded by fluid or lie in empty spaces; k, l, lumps of homogeneous material, surrounding two small blood-vessels, looking like the swollen Deiter's cells (from the section D IV-1); e, drops of fluid separating the nerve fibres in the oedematous white matter in D III-IV; m, n, partial liquefied Deiter's cells in the oedematous white matter in the section D IV-1. Some granular material lies about the nerve fibres near the cell n; x, y, very much swollen glia cells from the right posterior horn in the section D IV-1 (Fig. 5).

Fig. 5.—A portion of the section D IV-1 more highly magnified, showing the structure of the hyperplastic wall of the cavity, the oedematous condition of the gray and white matter and the distribution of the degenerating glia cells.

a. Inner zone of the wall, similar in structure to the limiting membrane; this inner zone is somewhat oedematous; it contains droplets of fluid and the interlacing glia fibres composing it are swollen and indistinct. b. Layer of fine filaments with small round nuclei wrapped circularly about the cavity. c. Layer of loosely packed small spider cells. d. Outermost compact layer of the wall, composed of filaments, granular matter, a few nerve fibres and very many liquefying glia cells (see Fig. 4, h, i). This outer zone at k involves the right column of Burdach where the filaments are more loosely arranged. f. Degenerated column of Goll, containing drops of fluid, degenerated Deiter's cells and cavities resulting from their liquefaction. e. Crescentic mass of fluid partially separating the wall of the cavity from the surrounding tissues. x. Cells of the central canal arranged in string-like rows. The gray matter and the white matter at h and it is honey-combed with drops of fluid which are represented by the white spaces

in the drawing. The small black spots represents the swollen and liquefied glia cells.

Fig. 6.—From a section of the tumor. Two small blood-vessels, invested by sheaths of scantily nucleated filamentous neuroglia, are surrounded by the tumor, which are so closely aggregated that the cell-bodies are not distinct.

Fig. 7.—A more highly magnified view of the section D VIII-2, showing the intra mural situation of the telangiectatic glioma sarcoma, and the structure of some of the nodules on the surface of the cord.

The thin layer, a, a, lining the tumor internally is identical in structure with the limiting membrane (*Fig. 2*). The tumor cells are grouped about the neuroglial sheaths of the numerous blood-vessels. The area of the tumor at c is loose-textured, and contains more neuroglia cells than sarcoma cells. At the left posterior root entrance an extension of the tumor forms one of the superficial nodules. The remaining surface-nodules in this section are polypoid outgrowths of the neuroglia of the white matter. The nodule, b, contains some small cavities apparently formed by the liquefaction of the glia cells, which are represented by the black spots in the gray matter and in the nodular gliomatous hyperplasias. The larger black spots correspond to the swollen glia cells. The nodules x, y, and z are growths of the neuroglia strands accompanying the nerve roots as they enter the cord.

VERTIGO AND STAGGERING IN TEMPORAL LOBE LESIONS.

REPORT OF TWO CASES OF FOCAL LESION OF THE RIGHT TEMPORAL LOBE.

By CHARLES L. DANA, M.D.,

NEW YORK.

Lesions of the temporal lobes often give rise to no localizing symptoms. In some cases, however, there are observed partial deafness of the opposite ear and disturbances of taste and smell.

When the lesion is on the left side and involves the posterior part of the first and part of the second temporal convolutions, word-deafness occurs. Hysterical symptoms have been noted in connection with tumors of the temporal lobes (Bramwell).

I have had under observation two cases of focal lesion of the right temporal lobe. In one case the lesion was well localized, chronic, and not especially irritative. Hence it furnishes an excellent pathological experiment in the study of localization.

The other case is much less valuable, but it is reported briefly for the reason that a similar type of symptoms was observed to that shown by the first patient.

In the first case there was a distinct hysterical condition present, and a peculiar class of forced movements. The patient had a frequent tendency to fall over to the right, and sometimes to whirl around to the right.

In the second case the patient also frequently fell over towards the right.

I have come across a third case, in which frequent staggering to the right side was a prominent symptom.

It has seemed to me, therefore, that possibly a close analysis of cases of lesions of this lobe might reveal this symptom often, and that it might have some diagnostic value. And this view might be justified physiologically by supposing that the vestibular branch of the auditory nerve, which has to do with our space sense, has a representation in the temporal lobe, as well as its companion, the cochlear root.

Physiologists have found that rolling and forced movements can be produced on animals by injuries of:

1. Cerebral cortex, especially in the parietal lobule.
2. Corpora striata.
3. Optic thalami.
4. Cerebral peduncles.
5. Pons Varolii.
6. Tubercular quadrigemina.
7. Cerebellar peduncles, especially middle.
8. Olivary bodies.

The principle places, however, are the olivary bodies, middle cerebellar peduncles, posterior tubercles of the corpora quadrigemina, and cerebral peduncles.

Extirpation of the temporal, parietal, or in fact any of the cerebral lobes in lower animals, has not systematically caused forced movements. This, however, does not prove that the cerebral cortex of man does not contain a representation of the sense of equilibrium or relations in space, but only that the function of equilibration may be carried on by other and lower centres.

Clinicians have observed forced movements in cases of lesion of the middle cerebellar peduncles and parietal lobes.

The only observations, so far as I know, bearing upon forced movements due to lesions of the cerebral hemispheres, are those collected by Nothnagel and Bechterew (*Arch. f. path. Anat. u. Physiol.*, 100 Bd. 3 H.). These consist of the cases of Romberg, Friedreich, Petrina, Mesnet, Longet, Romberg, Penzoldt, and Bechterew. Almost all of them are old cases, and will not stand close analysis. Bechterew, however, thinks that there is sufficient evidence

to justify the hypothesis that the anterior cerebellar peduncles are connected with the upper parietal lobe.

In my opinion, although there is evidence enough that this lobe has to do with the muscular sense, our space sensations are not disturbed by its irritation or destruction. The stumbling and awkwardness of ataxia may occur without vertigo or sense of confusion in our relations in space.

The fact which Schaefer has shown, that irritation of the tempero-sphenoidal lobe causes conjugate deviation of the head and eyes, and the fact that a large proportion of deaf-mutes cannot be made vertiginous, may be mentioned in this connection.

Besides this, Flechsig has described, and pathological cases reported by Bechterew, Flesch and Koneff, and Ros-solymo show, a tract of efferent fibres extending from the tempero-occipital lobes down through the outer part of the cerebral peduncles to the pons nuclei, thence connecting by the middle cerebellar peduncles with the cerebellum of the opposite side. Thus these lobes are placed by a band of efferent fibres in connection with the cerebellum.

CASE I. *Summary*.—Female, age 32. Blow on the head; eighteen months later, chill, fever, for three days, temporary rigidity of left arm, continuous occipito-frontal headaches, and vomiting; four months later, vertigo, forced movements, falling suddenly, always back and to right; short period of stupor and vomiting. Recovery, but constant attacks of falling toward right and backward, and of falling out of bed to right. Sudden fall and death. Duration of symptoms, two years.

Autopsy: focal encephalitis size of pigeon's egg in middle two-fourths of third and fourth temporal convolutions on right side. Recent hæmorrhage, bursting into lateral ventricle.

Detail.—Kate C., age thirty-two; mar.; Ireland; domestic, admitted to hospital September 29, 1888.

Family History.—Father died of dropsy, otherwise negative.

Previous History.—Always healthy. Has had rubeola and rheumatism for the past two winters. Denies

syphilitic taint; drinks moderately; menstruated first at fourteen years, always regular. No children; had a criminal abortion performed on a four-month foetus six years ago. Two years ago patient fell down an air-shaft, a distance of thirty-five feet, striking on her back and the back of her head. She was unconscious for several days; was in bed for several weeks. Her friends say she has always been different since, both in disposition, and has been "queer" in her mind. She has been at times ugly and destructive. Patient denies any epileptic history or any symptoms of epilepsy.

Present History.—Since her fall, was perfectly well until six months before admission, when she was taken with a chill, followed by fever, nausea, vomiting and anorexia, and was sick in bed for three days. Her left arm became stiff and she was unable to use it. This lasted only for a few hours. Since then patient says she has had frontal and sometime occipital headache almost continually, usually accompanied with nausea and often with vomiting. Three weeks ago patient suddenly became weak and dizzy and was unable to walk without falling. She was taken to the Harlem Hospital and remained there a week, during which time she was in a stupor. After recovery she was discharged. On the day of admission, while coming to hospital, patient became dizzy and fell as she was getting from the horse-car and struck her head, at the same time she had a severe chill and vomited considerably.

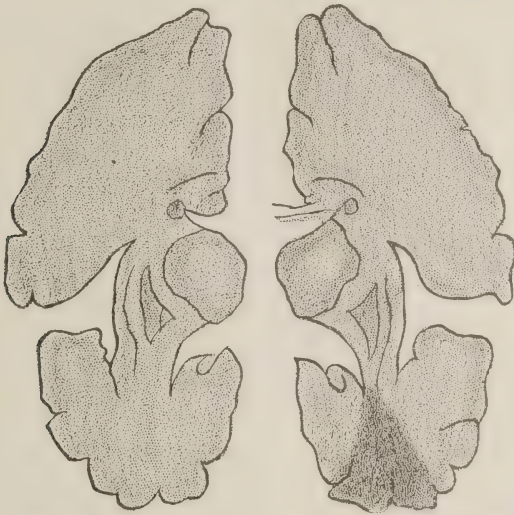
Physical Examination.—Patient is well nourished, not anæmic, tongue moist and slightly coated. Pulse, lungs, heart and liver are normal. Patient lies unconscious and will not rouse up. Occasionally, without apparent warning, she vomits spinach green matter in great gushes, ejecting it almost on the patient in the next bed. Over the right eye is a birth-mark, looking exactly as if patient had a black eye. Pulse, temperature and respiration were always normal. After being unconscious for two days, patient became semi-conscious, and complained of severe headache, which improved greatly under iodides, but finally this did not stop it, and an order was given for menthol., gr.

x, and antipyrin, gr. x, which never failed to stop it for the time being. This headache returned again and again, but was always relieved temporarily. Patient, as she grew more conscious, would be very restless, and fell out of bed continually always on the *right* side of the bed; finally she was tied in. After a few days she was up and about, even helping. She always walks with a stiff heavy tread and with her body bent backward and towards the right side, the body bent from the waist upward and appearing to balance and carry most of the weight on the right hip. Her speech was slow, deep-toned and very deliberate. She also would blurt out odd and amusing remarks. Occasionally, while carrying a pail of water or a dish, she would become dizzy and lose the power of holding what she was carrying, and once dropped hot water on her feet and scalded them quite severely. For the following three weeks after examination she had several attacks of unconsciousness lasting for two to three days each, during which she would vomit the same spinach green matter, and as she came out would always complain bitterly of the intense headache. About a week before she died she became subject to very severe attacks of dizziness, and would fall over backward *towards the right always*. These would come on suddenly and without warning, and patient would be standing upright and then would fall over on her head with such force as to cause her feet and legs to fly over beyond her head. Sometimes she would be found with her arm bent around an iron pillar in the centre of the ward, and swinging her body around it with great rapidity. Two days before she died, she continually threw herself out of the right side of the bed, and when sitting in a chair she would throw herself flat on her face. Finally, she started to walk across the ward and fell backward, striking her head with great violence. The nurse helped her into a chair in less than a minute. She stiffened herself out, and calling the nurse by name, she breathed a few times and died.

Autopsy showed every abdominal organ healthy; there were a few adhesions on the upper part of the right lobe of

the liver and at the base of the right lung ; otherwise lungs and heart were normal.

On removal of the brain, a good deal of blood was seen in the right middle fossa. On the under surface of the right temporal lobe was a focal lesion consisting of softened brain-tissue mixed with blood. The lesion involved the third and fourth temporal convolutions in their middle three-fourths, and communicated by a fresh opening made by the recent hæmorrhage into the right lateral ventricle. On carefully washing out the cavity, it was seen to be distinctly limited, as though the process of softening had been



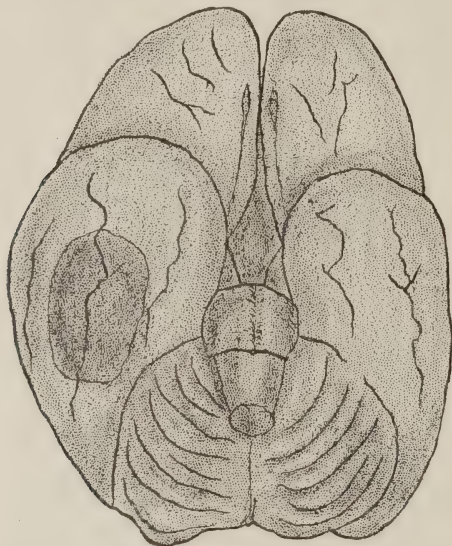
CASE I.—Abscess of Right Temporo-Sphencidal Lobe.
Hæmorrhage.

an old one. Microscopical sections of the limiting wall showed a decided inflammatory change, there being a thin pyogenic membrane evidently in process of forming. The point of lesion lay directly over the upper surface of the petrous bone, and at this point meninges were thickened and adherent ; but there was no communication with the ear, and on opening the mastoid cells and labyrinth no signs of inflammatory change were found.

The location and limits of the lesion are shown here.

The hippocampal gyrus was not involved.

The blood-vessels seen on microscopic section showed some thickening of the walls. Death was caused finally by a rupture of large vessel, which caused a hæmorrhage of sufficient magnitude to burst into the lateral ventricle. The lesion was, therefore, a focal encephalitis, having a slow course, and ended by a rupturing of a blood-vessel, which rupture might very well have been the result rather than the cause of her sudden fall.



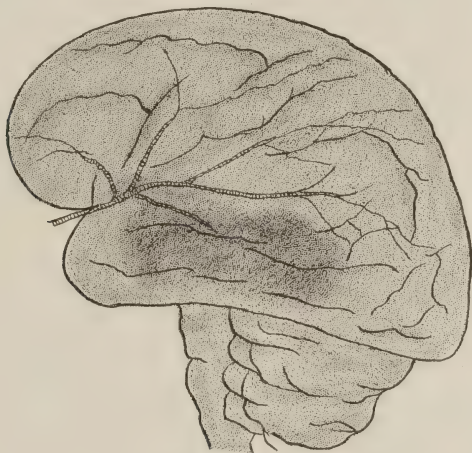
CASE I.—Abscess of Right Temporo-Sphenoidal Lobe.
Hæmorrhage.

An examination of the cerebral peduncles does not show a degenerated tract to the naked eye.

CASE II.—J. B. W., age 40. Patient was brought to the hospital delirious, and was put into the cell; he acknowledged drinking, and acted like a man in the active stage of delirium tremens. He gradually became unconscious in about twenty-four hours and died. His friends said he had had, a year ago, an attack of apoplexy; since then he has had several attacks of dizziness, and would fall forward, striking on the right side of his head; also he had had attacks of "twitchings." These appear to have been very severe and general; he would always fall in them,

and always injured the right side of his face, sometimes causing it to bleed. His memory previous to his apoplectic attack had begun to fail, and patient would have to be told facts several times before he would take them in.

Autopsy showed lungs in both lower lobes to be in state of red hepatization from pneumonia. Liver large and slightly cirrhotic. Heart normal. Kidneys, capsules adherent, cortex slightly increased, and markings a little indistinct. Brain: there was a great deal of serous effusion over the convexity, and there also appeared to be some beginning meningitis along the course of the vessels.



CASE II.—Chronic Meningo-Encephalitis. Right Temporal Lobe.

There was local pachymeningitis over the middle of the right second temporal convolution. On tearing away the membrane, a deep laceration of the brain at this point was made, and beneath it was seen a focus of softening involving the middle fourth of the second temporal superficially, extending deeply back and involving the second and third temporal as far as the occipital lobe, but not involving the ventricles. There was some recent meningo-encephalitic softening, also extending forwards and involving slightly the point of division of fissure of Sylvius and island of Rheil. The middle cerebral artery was not occluded,

but evidently one of its branches had become recently involved. The arteries were not atheromatous. Only one vertebrate artery could be discovered.

A case of abscess of the right temporo-sphenoidal lobe, with staggering to the right as a symptom, is briefly reported in the Providence Medical Journal, Jan., 1888.

In looking over the histories of the various lesions of the temporal lobes, I have not found any case, aside from the one just cited, which resembles those here reported, though several are recorded in which there were disturbance of motility.

The interpretation that I have put upon the symptoms is therefore justly open to criticism. I can only give the facts for what they are worth; and suggest that in the majority of cases the disease has been acute, in others a special examination of the vertigo and motor disturbance was not made.

It may be that the right temporal lobe is more especially related to the space-sense, just as the left lobe is more related to the sense of hearing.

I can affirm very positively that in this case there was no disease of the ear.

Finally, I would call attention to the well-known fact that the cortical representation of sensory organs is widely distributed. Destruction of one part is easily compensated for by other regions. One might easily expect that a central cerebral lesion irritating fibres from the vestibular nerve would usually cause only slight symptoms. It remains to be seen, therefore, whether *in future studies of lesions of the temporal lobes will not show that vertigo and staggering are not unusually frequent.*

50 WEST 46TH STREET.

A DESCRIPTION OF TWO CHINESE BRAINS.

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OUR knowledge of the racial characteristics of brains is as yet so limited that the following study of two Chinese brains will not prove uninteresting.

The first Chinese brain ever described was presented at a meeting of the American Neurological Association June 21, 1886, by Dr. Chas. K. Mills,¹ together with Dr. A. J. Parker, the latter having previously exhibited it at a meeting of the Philadelphia Neurological Society. In January, 1887, Dr. Mortiz Benedikt describes three additional Chinese brains.²

I propose briefly to describe the two specimens³ in my possession, and then to compare the results with those of the writers just mentioned. I will designate these brains as Number One and Number Two.

I. *Number one* was the brain of, as far as could be learned, a quiet and intelligent laundryman about twenty-five years of age. Death had been caused by phthisis.

The brain was large and well proportioned. Unfortunately it was not weighed. The orbital surfaces of the frontal lobes and basal surfaces of the temporals seemed more oblique or shelving than usual, i. e., these surfaces seemed slightly everted.

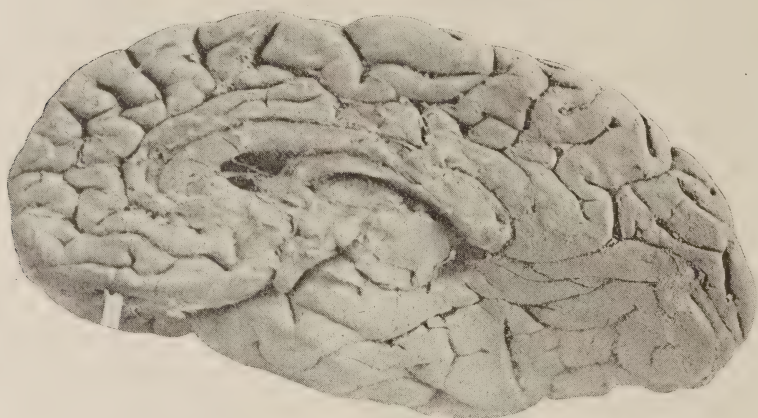
Right hemisphere.—The Sylvian fissure is decidedly

¹ Preliminary Study of a Chinese Brain. By A. J. Parker and Chas. K. Mills. JOURNAL OF NERVOUS AND MENTAL DISEASE, Vol. xiii., No. 10, 1886. (Embodied in the Address of Dr. Mills.)

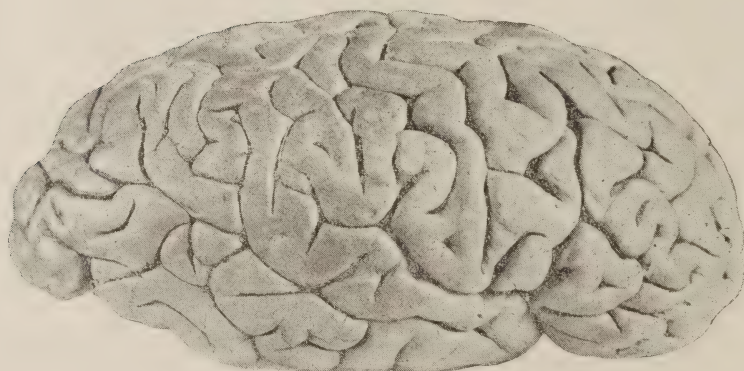
² Drei Chinesen Gehirne. Prof. Dr. Moriz Benedikt. Medizinische Jahrb., 1887.

³ I am indebted for the opportunity of studying these brains to Dr. H. F. Formad, of Philadelphia.

short, though a superficial confluence greatly increases its apparent length. Its direction is average. Its anterior branch is confluent with the precentral, in fact constitutes with the latter one deep fissure. The horizontal branch terminates in two short branches one perpendicular and the other horizontal in direction. The fissure of Rolando is



BRAIN NO. I.—Right Half.



BRAIN NO. I.—Right Half.

average in direction, but decidedly sinuous, though not more so than is occasionally observed in some white brains. It is all but confluent with the Sylvian fissure, the operculum being here reduced to but little more than an eighth of an inch in width. Its upper end extends well into the medial surface. In its lower third a shallow fissure makes

it confluent with the precentral which at this point is somewhat irregular. The first frontal fissure is very sinuous and is confluent with a superior precentral, the latter being long and deep. A well-developed and sinuous medi-frontal is present, and is, together with the second frontal fissure which presents nothing remarkable, confluent with the precentral.

Immediately back of the fissure of Rolando is found an exceedingly sinuous retro-central. It runs quite parallel with the central except that it exaggerates the sinuosities of the latter. It arises close to the Sylvian and extends fairly up to the mesial edge of the hemisphere. In its middle portion it becomes confluent by a short transverse fissure with the anterior portion of the interparietal. The latter takes its origin in the gyrus included by the terminal bifurcation of the Sylvian, and ascends in an average direction but exceedingly sinuous course, and finally terminates in a very large and deep transverse occipital. It is not confluent with the parieto-occipital, though a deep and well marked external perpendicular fissure intersects it.

The parallel fissure is of unusual length and presents many confluences. It extends backward in an average direction, with but few shallow interruptions. Just back of the termination of the horizontal ramus of the Sylvian it gives off a well-marked perpendicular branch. A little farther on it becomes confluent with a more or less well-marked fissure of Wernicke. Finally it joins the transverse occipital, and then terminates in a fissure immediately below and parallel to this. This last mentioned fissure is very large and deep and looks like a vegetative repetition or reduplication of the normal transverse fissure. A deep notch at the basal edge of the occipital lobe forms an indication of a second Wernicke.

The mesial surface gives one the same general impression of complexity as does the lateral surfaces. The colosso-marginal fissure pursues an average course, but extends over half an inch on the lateral surface. It gives off numerous and irregular perpendicular branches, and the mesial surface of the first frontal convolution is made very

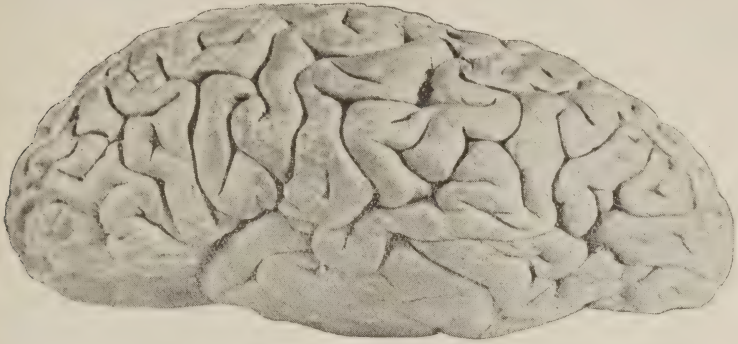
complex by their 4-shaped and b-shaped fissures. The paracentral lobule is divided transversely by a very deep and long fissure. The quadrate lobule, which is exceedingly large, is also rendered very complex by an abundance of transverse and perpendicular intersecting fissures. The parieto-occipital fissure is very sinuous, and enters directly into the hippocampal fissure, the gyrus fornicatus being entirely submerged. The calcarine is short and curved very strongly upwards. Its posterior termination is simple, i. e., no bifurcation is present. The cuneus is quite small and is made very complex by deep transverse fissuration. The lobulus fusiformis and lobulus lingualis are separated by a sinuous collateral and present in addition numerous and confluent secondary fissures.

Left hemisphere.—The Sylvian fissure is a little longer than its fellow of the opposite side. Its anterior branch is normal in appearance and not confluent with the precentral, as in the other hemisphere. The horizontal ramus again terminates in two short bifurcations. The fissure of Rolando is excessively sinuous and is *directly confluent with the Sylvian*. Its upper end extends fairly to the mesial edge of the hemisphere. The first and second frontal fissures are less sinuous than in the other hemisphere. The former is confluent with a long and deep precentral, while the latter is confluent with a smaller perpendicular fissure in advance. As in the other hemisphere a long and sinuous retro-central is present. It is, like the central, confluent with the Sylvian. The interparietal pursues an average direction, but its anterior and posterior portions are distinctly separated, and further, it fails to terminate in (i. e., become confluent with) the transverse occipital. As in the other hemisphere this last mentioned fissure is exceedingly deep and long.

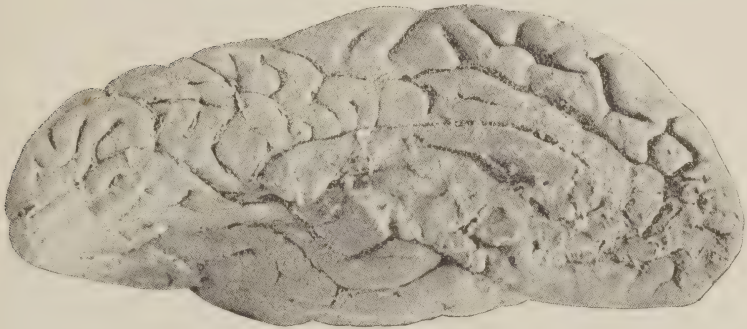
The parallel fissure, contrary to its fellow, is short ; it is also exceedingly sinuous. In a direct line however with its course, we find a fissure confluent with a number of irregular fissures, among which are two perpendiculars. No vegetative repetition of the transverse occipital is present.

The mesial surface closely resembles that of the other

hemisphere. The callosa-marginal is very long and incises the lateral surface to the depth of fully an inch. The mesial surface of the first frontal though much marked by second-



BRAIN NO. I.—Left Half.



BRAIN NO. I.—Left Half.

ary fisuration is less so than its fellow. The quadrate lobule bears a close resemblance to that of the other side. The parieto-occipital fissure is again very sinuous and again terminates directly in the hippocampal fissure. The gyrus fornicatus is not however wholly submerged. Further the parieto-occipital extends to an extraordinary degree upon the outer surface, namely, over an inch. A well defined *pli de passage inférieure externe* is however present. The calcarine fissure is as before strongly curved upward, and simple in its termination. The cuneus though small is larger than that

upon the opposite side. Its surface bears an irregular secondary fissure.

The collateral fissure resembles closely its fellow. Secondary fissuration of the lobulus fusiformis and the lobulus lingualis is not however as marked.

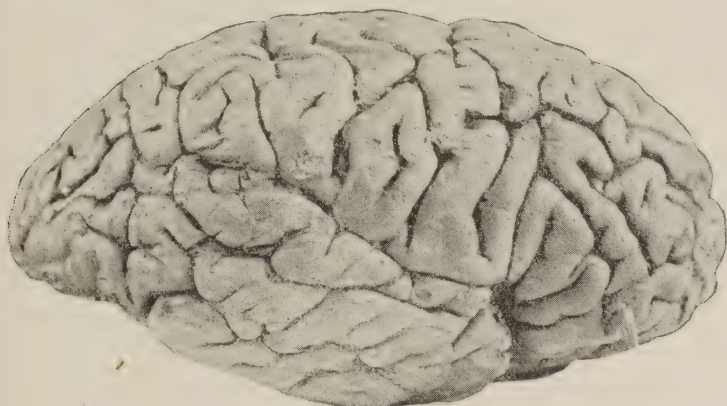
The orbital surfaces of both frontal lobes bear deep *h*-shaped fissures more complex in the right than in the left hemisphere.

II. *Number two* was the brain of a dissipated china-man, aged about thirty-five, who was found dead in an opium den.

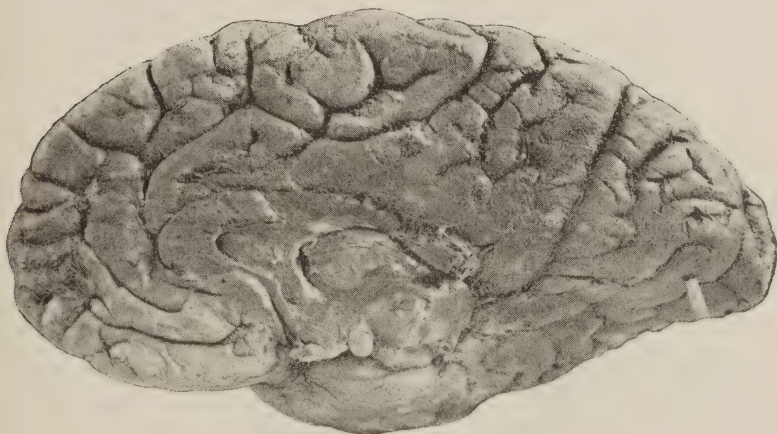
The brain is large and also well-proportioned. The orbital and basi-temporal surfaces likewise exhibit the oblique character noted in *Number one*.

Right hemisphere.—The Sylvian fissure is average in course and direction, though of unusual length. The fissure of Rolando is exceedingly sinuous. It is all but confluent with the Sylvian, being separated by a very narrow bridge. It extends fairly up to the mesial edge. The first frontal fissure does not begin in its usual position but at what would correspond to a junction of its lower and middle thirds. It is sinuous and interrupted, and finally terminates in an upper deep precentral. A deep and well-marked fissure runs from the mesial surface transversely across the frontal lobe immediately below the origin of the first frontal. A second fissure arises from the outer edge of the orbital surface, passes at first perpendicularly upward, joins the transverse fissure just described and thence follows the average course of the medi-frontal. By means of these fissures the tip of the frontal lobe is converted into a rectangular lobule, the surface of which still further presents well-marked transverse fissuration. The medi-frontal terminates in a large precentral. The third frontal is short and terminates in a perpendicular fissure which is parallel to and slightly in advance of the normal precentral. There are thus present in this hemisphere two precentral fissures, the most interior being a reduplication or vegetative repetition of the normal sulcus. This adventitious precentral, besides receiving the third frontal also intersects the medi-

frontal and is directly confluent with the Sylvian at a point posterior to the anterior ramus of the latter. The frontal lobe is made still further complex by the presence of a short fissure parallel to the third frontal and between it and the medi-frontal. Its inferior end is confluent with a shallow transverse fissure running into the Sylvian.



BRAIN No. II.—Right Half.



BRAIN No. II.—Right Half.

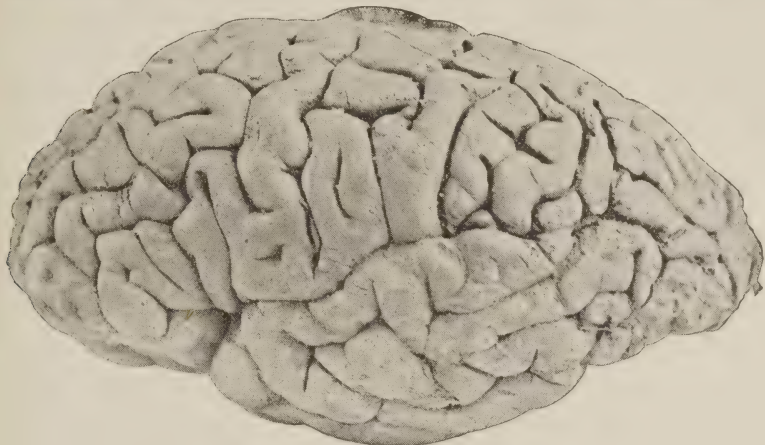
The interparietal has its origin in the Sylvian with which it is fairly confluent. After proceeding a short distance it sends a communicating branch to the fissure of Rolando, and a little farther on gives off a short retrocentral branch parallel to the latter. In its posterior portion it becomes fluent with the parieto-occipital, the *pli de passage enferieur*

externe being entirely wanting. It finally terminates in a short transverse occipital. The parallel fissure is as in brain *Number one*, right hemisphere, exceedingly long, and finally becomes confluent with a typical external perpendicular fissure. Shortly in advance of this perpendicular fissure is found another, having the same general direction and intersecting the inter-parietal. Numerous other secondary fissures and markings having the same general direction are found. A vegetative repetition of the transverse occipital is also present. It exists directly below the normal fissure, is long and deep and extends over the mesial edge far into the cuneus.

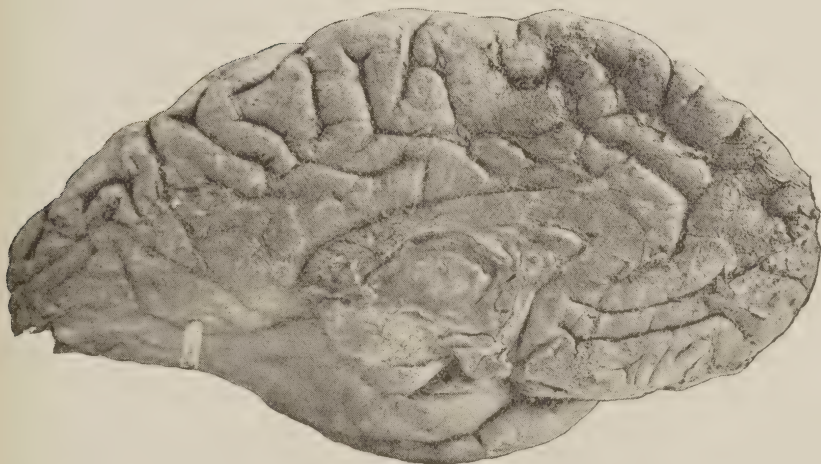
The mesial surface presents, to begin with, a deep and very sinuous calloso-marginal. It gives off numerous perpendicular and irregular branches, especially into the paracentral lobule, and thence terminates freely upon the lateral surface. The quadrate lobule presents quite a number of characteristic fissures, but is not otherwise remarkable. The parieto-occipital fissure is very straight and as in the hemispheres of brain *Number one*, is directly confluent with the hippocampal fissure, the gyrus fornicatus being, as before, submerged. The calcarine presents nothing remarkable, its course being average and its posterior end being bifurcated. The cuneus is deeply incised by the adventitious transverse occipital already described. The collateral presents nothing worthy of note other than confluence with numerous transverse fissures.

Left Hemisphere.—The Sylvian fissure is decidedly sinuous, and, omitting a minute and shallow interruption, of excessive length. Its termination is vertical and slightly recurrent. The fissure of Rolando is average in course and direction. Like in the other hemisphere, it is separated from the Sylvian by an exceedingly narrow gyrus. The first and second frontal convolutions present an average appearance but are both confluent with the upper segment of the precentral, which is very long and well developed. A short medi-frontal is present. The lower segment of the precentral is comparatively small and not confluent with any of the frontal fissures, but directly so with the Sylvian

The excessive transverse fissuration so noticeable in the right frontal lobe is not evident here. A well-marked and deep retrocentral fissure is present. It arises directly from the Sylvian, and early in its course communicates by a horizontal branch with the central. Then, after a slight



BRAIN NO. II.—Left Half.



BRAIN NO. II.—Left Half.

interruption, it passes upward parallel with the central, and thence over the mesial edge for nearly an inch. The interparietal fissure likewise springs from the Sylvian, and like the retro-central is early interrupted by a narrow convolu-

tion. Thence it passes backward in an average direction but fails to terminate in the transverse occipital. Like its fellow of the opposite hemisphere, it also is confluent with the parieto-occipital, the *pli de passage superieur externe*, being as before, entirely absent. An external perpendicular fissure is likewise present, though it does not intersect the inter-parietal, as in the other hemisphere. The parallel fissure at first interrupted in its course becomes at its posterior extremity confluent with the external perpendicular fissure just mentioned. The transverse occipital is small and no reduplication of it exists. Two anomalous fissures running in a longitudinal direction from the apex of the occipital lobe forwards, join the external perpendicular.

The colosso-marginal is not very sinuous. A marked tendency to repetition is found near its origin and also in the middle third of its course. It ends upon the mesial surface and in one or two places becomes quite shallow. The paracentral lobule bears an irregular *h*-shaped fissure. The quadrate lobule is large and its fissures numerous and deep. The parieto-occipital is long and straight, and as in the other hemispheres described, terminates directly in the hippocampal fissure, the gyrus fornicatus being as before submerged. The calcarine is average in course and direction. Its posterior extremity is bifurcated. The cuneus bears a small though deep fissure having sharp upward curve and presenting no confluence. The collateral presents nothing worthy of note. However, a deep and anomalous fissure extends from the apex of the temporal lobe just external to the uncinate gyrus in an oblique course backward and outward to the lateral surface, which it deeply incises at a point midway between the apex of the temporal and the apex of the occipital lobe. Its presence gives the base of the temporal lobe a very unusual appearance.

Comparing now, these results with those of the previous observers, we find in the first place, that the obliquity or eversion of the obital surfaces spoken of by Mills and Parker is also very noticeable in both of these brains. Benedikt observed this fact independently of Mills and Parker in all three of his brains, and in addition stated that the basi-

temporal surfaces also partook of this quality. In the brains I have just described the same fact obtains. In brain *Number two* indeed the lateral surface of the temporal lobes is markedly increased by this eversion, the basal surface being narrowed down to little more than the hippocampal and uncinate gyri. In brain *Number one* the eversion is not marked at the time of writing, the brain having been somewhat distorted during the process of hardening, but it is evidently present.

This eversion of the basal surfaces is very significant, and having been observed in the six instances thus far studied seems to deserve the rank of a racial characteristic.

Mills and Parker mention in addition to various other minor peculiarities the extreme length and confluence of the parallel fissure which is noticeable also in both of my own specimens. They note also unusual complexity of the colosso-marginal of one side and instances of vegetative repetition in both ; points which these brains likewise show.

The Sylvian fissure is decidedly long in one of the hemispheres of Mills and Parker and very long in both hemispheres of *Number two* of my own specimens. Benedikt makes no especial mention of the relations of the Sylvian fissure, and it is somewhat difficult to judge from his drawings. However, I take it that very unusual length is not characteristic of the fossa in his brains.

The fissure of Rolando in the brain of Mills and Parker, judging from the photograph, was in both hemispheres very nearly confluent with the Sylvian. In Benedikt's brains, judging from the drawings, the same condition was present in the right hemisphere of his first brain, in both hemispheres of his second, and in the left hemisphere of his third brain. The same condition obtains in the right hemisphere of brain *Number one* of my own specimens, and in both hemispheres of brain *Number two*. Again, in the right hemisphere of Benedikt's third brain the central fissure is directly confluent with the Sylvian, This also obtains in the right hemisphere in brain *Number one*.

In the brain of Mills and Parker the inter-parietal presents nothing beyond a division by a bridging convolu-

tion into two parts. It was not confluent with the parieto-occipital. In the drawings of Benedikt, however, it is seen to be confluent with this fissure in three instances, twice in the same brain, the third, and once in the second. In my own specimens this confluence exists twice, namely: in both hemispheres of brain *Number two*. External perpendicular fissures are present in the brain of Mills and Parker, in all of my own specimens and apparently in some of Benedikt's.

Again, in the Mills-Parker brain, the parieto-occipital bears in the left hemisphere a normal relation to the hippocampal, i. e. it does not become confluent with the hippocampal, and the gyrus fornicatus is at this point fully up to the brain level. In the right hemisphere this confluence all but takes place, the gyrus fornicatus being reduced to a mere shred. In the brains of Benedikt this confluence is depicted as taking place twice, i. e., in both hemispheres of his first brain. In my own specimens this confluence takes place in every instance, the gyrus fornicatus being always completely submerged.

Benedikt states also that in the right hemisphere of his second brain the connection between the calcarine and the parieto-occipital is wanting. However, his drawing shows just the opposite, the normal confluence between these fissures being depicted. It is unfortunate that the drawing and the text do not agree, as this observation would be of extreme interest.

Further Benedikt remarks that the temporal and occipital lobes can each be very readily separated into four convolutions or lobules. This is not evident in the brain of Mills and Parker, though in my own specimens the temporal lobe can after some reflection be arranged into four parts; but this does not hold good of the occipital.

Benedikt also states that the central fissure is very sinuous and tends to confluence with the precentral or retrocentral. This observation can to a great extent be corroborated by my my own specimens and that of Mills and Parker.

In attempting to generalize from an analysis of these

brains we must be exceedingly cautious. Nevertheless, these striking peculiarities appear to be present : They are, first, the eversion of the orbital and basi-temporal surfaces; secondly, the extreme senuosity of the fissures; thirdly, the unusual confflexity, if not the unusual size of the frontal lobes. Regarding the excessive transverse fissuration, it may be remarked that it is also found in white brains, though far less frequently; and as regards the unusual confluences of fissures, we can certainly say that they are relatively rare in the white brain and very frequent here, and this becomes of some significance perhaps, when we recall that these conditions are very frequent in the brains of negroes, and largely obtain in the brains of the apes. This is especially true of the confluence between the parieto-occipital and the inter-parietal, that between the parieto-occipital and the hippocampal, the presence of a deep and well-differentiated external perpendicular and the consequent absence of the various external *plis de passage*.

A NOTE ON THE "PLI DE PASSAGE INFÉRIEUR INTERNE" IN THE HUMAN BRAIN.

By F. X. DERCUM, M. D.,

Instructor in Nervous Diseases, University of Pennsylvania, Neurologist to Philadelphia Hospital.

IN an examination of thirty-three negro brains A. J. Parker¹ discovered two instances in which no confluence occurred between the parieto-occipital and the calcarine fissure. That is, they were cases in which the inferior internal *pli de passage*² had developed fully up to the general level of the brain. Up to this time the development of this convolution had been unknown in the human brain, though its existence in the apes is constant with the exception of *Hylobates* and *Ateles*. In the white brain it has since been found, though developed in a very imperfect degree by Dr. Charles K. Mills,³ in the brain of Joseph Taylor, exhibited by him in his presidential address in 1886. Benedikt has also demonstrated the brain of a criminal in which the calcarine was separated from the parieto-occipital, and he describes a similar condition as occurring in a Chinese brain. Unfortunately, however, the accompanying drawing depicts exactly the normal condition, *i. e.*, the confluence of the two fissures.

It recently occurred to me to search for this convolution among idiot brains, and accordingly I availed myself of Dr. Wilmarth's kind invitation to examine his collection of idiot brains at the Institution for Feeble Minded Children at Elwyn, Pa. Here, out of seventy-five brains I found four instances. Two of these existed in opposite hemispheres

¹ Proceedings Acad. Nat. Sci., Philadelphia, 1878. Cerebral Convulsions of the Negro Brain, p. 11, and Simian Characters in Negro Brains, p. 339.

² Gratiolet. Gyrus cunai of Ecker.

³ Arrested and Aberrant Development of Fissures and Gyris in the Brains of Paranoiacs, Criminals, Idiots and Negroes, Etc., by Chas. K. Mills, JOURNAL OF NERVOUS AND MENTAL DISEASE, 1886, Nos. 9 and 10.

of the same brain. In every instance the convolution was large and well developed.

In addition it was twice found (in the same brain) barely submerged.

All of these brains in which the convolution occurred were those of white low grade idiots.

The accompanying sketches represent the condition found.



1b Right Hemisphere.



1a. Left Hemisphere.



Fig. 2.



Fig. 3.

THE RELATION OF THE THALAMUS TO THE PARACELLE (LATERAL VENTRICLE).

By BURT G. WILDER, M.D.,

Professor of Physiology, Comparative Anatomy, and Zoology, Cornell University, Ithaca, N. Y.

UPON two previous occasions—in the Cartwright Lectures for 1884 (New York Medical Journal, April 26, 1884, pp. 460-461), and in the course of discussion of my paper, "Notes on the Brain," read before this association in 1886—I called attention to the inadequacy of standard anatomical manuals with regard to the circumscription of the encephalic cavities and particularly the vagueness and inaccuracy of all figures purporting to exhibit the relation of the thalami to the strictly cerebral division of that cavity.

The objects of the present communication are these :

1. To show that my criticism is still applicable, even to publications of later date.
2. To indicate points which require further observation.
3. To suggest modifications in the modes of preparing, dissecting, describing and figuring brains which are intended to convey information respecting the subject under consideration.

The following terms may not be familiar to all, and are briefly defined.

Fimbria, the fibrous margin of the fornix, often called *corpus fimbriatum*.

Tænia, the fibrous band often called *tænia semicircularis*.

Rima, the line or area of interruption of the proper nervous parietes of the cavities for the intrusion of the plexus ; one of its margins is the *tænia*, and the other is the *fimbria*.

Rima is not synonymous with "great transverse fissure," since the latter, as well stated by Quain (II., 349), is an artificial condition produced by pulling out the plexus and tearing the membranes along the margins of the tænia and fimbria.

Ripa, any line of reflexion of the endyma upon a plexus or upon the pia. When the parts are separated, the torn or cut margin of the endyma may be traced as a more or less distinct ridge.

Ectocælian, outside the encephalic cavity; extra ventricular.

Entocælian, located or appearing within the encephalic cavity; intra-ventricular. Entocælian surfaces are always covered by endyma; ectocælian surfaces are commonly covered by pia.

Striato-thalamic groove, the "sulcus limitans" of Schwalbe; the furrow between the thalamus and the caudatum, the caudate or entocælian division of the striatum.

Plexal-groove, the "*sulcus choroideus*" of Schwalbe; the shallow furrow on the dorsal surface of the thalamus corresponding with the margin of the fimbria and the course of the plexus.

Diacæle, the proper cavity of the diencephal or thalamic segment of the brain; it corresponds to the "third ventricle less the aula which belongs to the prosencephal.

Prosocæle, the entire cavity of the prosencephal or fore-brain; including the paracæles or "lateral ventricles," the portæ or "foramina of Monro," and the aula, the mesal space between them.

Paracæle, either lateral division of the prosocæle; a right or left "lateral ventricle."

This is the only new term. It is proposed after several months consideration, as a substitute for procæle. I have used procæle at different periods, both for the entire prosencephalic cavity, and for its lateral portions; it has not been adopted by others, and is hereby withdrawn; *paracæle* conveys the idea of laterality and is not likely to be mistaken for *prosocæle*, the whole, of which each paracæle is a part.

I. Coming now to the three proper subjects of this paper, I have to reiterate my former criticism as to the way in which the paracœlian relations of the thalamus are represented in professedly anatomical treatises, ancient and modern.

Purely histological, embryological and physiological publications are not here included; nor those which have no figures. Mihalkorics and Hadlich distinctly contravene the current statement that the thalamus forms part of the floor of the "lateral ventricle," and the peculiar relations of the entire cerebrum to the thalami are so admirably summarized by Spitzka (*Medical Record*, July 26, 1884, p. 111) that had the article been accompanied by an illustration, one of the reasons for preparing the present paper might not have existed.

The works consulted are by the following anatomists:

Harrison Allen, Bourguery and Jacob, Burdach, Dalton, Darling and Ranney, Edinger, Gegenbauer, Gray, Huguenin, Henle, Mendel, Meynert, Quain, Sappey, Schwalbe, Viegdazyr, Weisse.

In some of these treatises the text may contain partial statements of the truth; but the very conditions which render a good figure more instructive than a description, cause illustrative imperfections to outweigh excellences of text; hence Quain and Schwalbe, for example, are to be judged not by their words, but by their more effective pictures.

Admitting, for the sake of occupying common ground, that a certain area of the dorsal surface of the thalamus is covered by endyma; that it is continuous with the caudatum, and that therefore, like that body, it enters into the composition of the paracœlian floor, none will deny an adjoining area of this same dorsal surface is as distinctly covered by pia; that it is continuous with the optic lobes, and like them wholly excluded from the encephalic cavity.

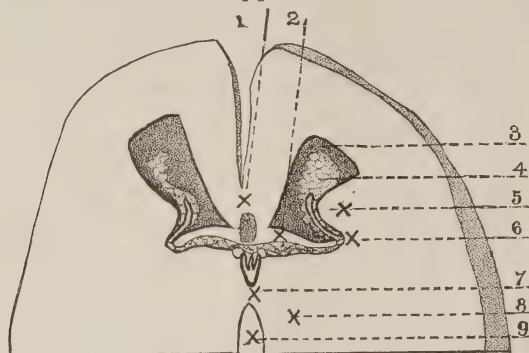
To represent the entire dorsal aspect of the thalamus as a smooth, unbroken surface is practically to affirm one or two things; either the whole is pial or ectocœlian, which would be in contravention of universal belief; or else the whole is endymal or entocœlian, which would involve not

only the optic lobes but the cerebellum and oblongata, a manifest *reductio ad absurdum*.

This, nevertheless, with perhaps a single partial exception, is what we find in the treatises above enumerated. The figures, whether representing natural surfaces or sections, fail to exhibit any lines of demarcation between entocœlian or ectocœlian areas either by a difference in texture or by raised edges to indicate where the reflected endyma had been divided. The exception referred to is Meynert's "Psychiatry," where Fig. 16 includes a line marked L, crossing the thalamus, and more distinct upon the left than upon the right; upon the explanation it is named "Linea aspera," but there is no further reference to it, and its morphological significance is not mentioned; consequently, although it probably does represent a torn edge of endyma, the figure alone would not convey any definite information. The extreme limits of misrepresentation are reached in Huguenin (Fig. 49) where the utter absence of distinction between surfaces which are said to be inside, and other surfaces which are said to be outside, can hardly fail to mislead or at least confuse the reader who is not already thoroughly grounded in correct notions as to the general morphology of the brain.

II. In the effort to prepare accurate drawings and a complete description of the paracœlian relations of the human thalamus, I have encountered certain unexpected difficulties which can be removed only by fresh preparations not now available. I confine myself therefore on the present occasion to pointing out a few matters which require further scrutiny. Among these are the existence, nature and significance of the seldom-mentioned band called by Vicq d'Azyr the *lamina cornea*; the extent of the really endymal surface of the thalamus; the relation of the pia and the endyma to the adjoining area where the latter is not adherent; the existence and extent of any paracœlian surface of the thalamus in anthropoid apes; the age at which in the human brain, the margins of the rima, the tænia and the fimbria, diverge to assume the adult condition; the accompanying figure shows that in a fœtus estimated at four

months, these parts are no farther from one another than in the lower mammals, where, even in hydrocephalus, as I showed in my first communication to the association in 1882, and as appears in the accompanying figure of a dog's brain, there is barely room for the intrusion of the plexus, and the thalamus does not appear at all within the paracœle.



TRANSECTION OF A FŒTAL BRAIN.

1. Callosum. 2. Fornix. 3. Paracœle. 4. Plexus. 5. Caudatum. 6. Rima. 7. Medicommissure. 8. Thalamus. 9. Diacœle.

1. The medicommissure is perfectly distinct and well preserved.

2. The paracœles (lateral ventricles) are higher than wide, and half their height lies dorsad of the level of the callosum.

3. The caudatum (caudate portion of the striatum) forms a marked projection of the lateral wall.

4. The fornix at this level occupies about one-third of the entire width of the cerebrum, whereas in the adult it measures not more than one-fifth, and probably more nearly one-seventh.

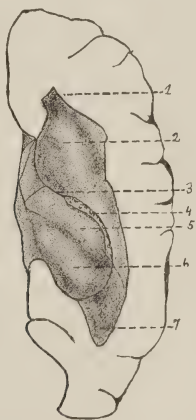
5. The fimbria, constituting the margin of the fornix, reaches the striato-thalamic groove, which demarcates the thalamus from the striatum, the diencephal from the prosencephal. In other words, each hemifornix is equal in width to the corresponding thalamus.

6. As corollaries to the preceding: (*A*) the dorsal surface of the thalamus is wholly pial, with no endymal portion as in the adult; (*B*) the thalamus does not, in any sense,

enter into the composition of the paracælian floor. This point is discussed at some length in my paper, 56, pp. 460, 461.

It will be seen that most of the foregoing points are more or less distinctly related to the increase in width of the entire brain, and specifically to the lateral extension of the thalami during the later stages of fœtal life.

The non-appearance of the thalamus in the paracæle, and, the concomitant narrowness of the rima, are also significant, because it is the permanent condition in all other mammals with the possible exception of some other primates. Hence these facts bear upon the important question as to the nature, extent, and significance of human or primatial peculiarities.



RIGHT HEMICEREBRUM OF HYDROCEPHALUS DOG.

1. Præcornu. 2. Striatum. 3. Rima. 4. Plexus. 5. Fornix.
6. Hippocampus. 7. Postcornu.

Notwithstanding the distension which has produced a postcornu, the rima is not widened, and the thalamus is wholly excluded from the cavity.

III. Pending a full account of the matter in the future, by some other anatomist if not by myself, there are some suggestions that may properly be made in connection with it.

Brains prepared in the usual way by alcohol or chromic acid liquids are not well adapted to accurate determination of the relations and attachments of the parts concerned ; either the membranes become detached or the masses are ill-preserved, or the cavities are practically obliterated. By continuous alinjection (injection of alcohol) either into the arteries or into the cavities, or both, there are secured perfect adhesion of the membranes and plexus, firmness of the nervous tissue, and a normal separation of the cœlian parietes from one another. Instead of trying to retain the entire cerebrum, it is better to study the paracœlian relations of the thalamus upon a mass containing little more than the parts concerned, which are then less apt to be torn or broken in handling.

The investigator should bear in mind that all the relations are complicated by the over-lapping of the encephalic segments ; by the curvatures and twistings of the parts ; and by the variations in width of the rima, or interval between the tænia and the fimbria.

The anatomical teacher and writer should lay stress upon certain fundamental ideas respecting the brain ; that the organ consists of a series of segments, some of which are greatly overlapped by others ; that the neurocœle, the cavity of the neuraxis, is completely circumscribed excepting for the so-called "Foramen of Magendie ;" that the mesal cavities, ending with the diacœle, communicate with the lateral cavities, the paracœles only through the aula and the portæ or "foramina of Monro ;" that in all other mammals, excepting perhaps the anthropoids, the fornix is wide and the rima narrow, the thalamus being wholly excluded from the paracœle ; that, finally every entocœlian surface is covered by endyma, and that any interruption of endymal continuity should be distinctly indicated.

In anatomical treatises the thalami should be described and figured in connection with the other constituents of the diencephal, and only incidentally as a quasi-member of the prosencephal.

In every such treatise there should be at least one representation of a transection, in which, upon a large scale

made possible by the omission of the dorsal and lateral regions of the cerebrum, there may be shown the unbroken continuity of the paracœlian endyma from the caudatum to the hippocamp, over the tænia, the plexus, and the fimbria. In a second figure, after removal of the paracœlian roof, its floor should exhibit the same parts in their natural and undisturbed relations, the endymal surface uninterrupted and the pial surfaces wholly excluded from the cavities. In a third the fornix, velum and plexuses might be removed ; but then the dorsal surface of the thalamus should present two distinct sharp edges, each a “ *linea aspera* ” of Meynert, a *ripa* Of the present writer. One of these would run parallel with the habena and demarcate the dorsal and pial surface of the thalamus from its mesal or diocœlian surface ; the other would cross the dorsal surface obliquely caudo-lateral, and demarcate the larger pial area from that comparatively small area which is continuous with the tænia and caudatum ; this figure should also exhibit distinctly the two grooves already named, the striato-thalamic and the plexal.

Society Reports.

AMERICAN NEUROLOGICAL ASSOCIATION.

Meeting June 26th, 1889.

A DESCRIPTION OF TWO CHINESE BRAINS, AND A NOTE ON THE PLI DE PASSAGE INFÉRIEURE IN THE HUMAN BRAIN.

Dr. F. X. DERECUM, of Philadelphia, presented two contributions with these titles. For original papers, see page 421.

He exhibited the brains described. Dr. Mills had made a morphological analysis of one in 1886, and descriptions of three Chinese brains had been added to literature by Moritz Benedikt. The six brains thus far analyzed exhibited unusual complexity due to excessive sinuosity of the gyri, and a tendency to excessive transverse fissuration. The frontal lobes were especially large and complex. There was unusual confluence of fissures, indicative of a low degree of development, such as was often seen in the negro's and sometimes in the white man's brain. There was unusual length of the parallel and Sylvian fissures, also eversion of the orbital and temporal lobes.

Dr. MILLS said it was very important to keep adding to our morphological descriptions of such brains until accurate deductions could be drawn from a very large number.

Dr. SPITZKA stated that the dictum that a tendency to confluence of fissures indicated a low type of development was not accepted by most anthropologists. The more brachycephalic the skull, the greater was the development of transverse secondary folds. The elephant, which stood among the highest of mammals, had a broad skull and a tendency to confluence of fissure. The Chinese were not

low in development. The internal *pli de passage* in the negro and idiot was significant only when the general cortical development was poor. The real cause of the location of the chief sulci lay in the inherent architecture of the brain, and was due to the arrangement of groups of cells and fibres, but the secondary and tertiary sulci might be influenced by other factors, such as the course of vessels. While we might never be able to localize the higher functions of the mind, there was something in the physiognomy of the Chinese brain which struck him as differing altogether from that of the Caucasian. But it was not the same difference as was noticeable between the negro and Caucasian brains. The peculiar moral attitude of the Chinese pointed to some relation between the structure of their brains and the character of their minds. Still, he had observed a great difference between these two Chinese brains, and there was no feature in them which might not be reproduced in the Caucasian brain.

Dr. DERCUM had not wished to imply that transverse fissuration was indicative of a low type, but thought the unusual confluence showed a lack of cortical development. He did not consider them brains of a low type, but many features such as were found in the brains of the negro and ape were found here also. The excessive sinuosity, however, indicated a higher cortical development. For instance, in one brain there were five well-developed frontal gyri.

Dr. SPITZKA had the same idea as Dr. Dercum. As compared with the Caucasian brain, the Chinese was in some respects inferior, and in others exhibited a superabundance of gyri. He spoke of the resemblance between the negro and ape brains. The difference between the brains of the orang-outang and chimpanzee were greater than those between the brains of the chimpanzee and a human being. There was no uniform appearance of the ape's brain. Vogt and another had classified the human brain into three types—the negro, Mongolian, and Caucasian—corresponding somewhat to the three lower types of the gorilla, chimpanzee, and orang-outang.

Dr. M. Allen Starr, of New York, then read a paper entitled

THE PATHOLOGY OF SENSORY APHASIA, WITH AN ANALYSIS
OF FIFTY CASES IN WHICH BROCA'S CENTRE
WAS NOT DISEASED.¹

In questions regarding the localization of cerebral functions, the final appeal must always be made to pathology. And since it is the practical application of the principle to the removal of disease from the human brain which is the chief object in view, its paramount importance cannot be denied. The collection and analysis of reliable and well-observed cases of disease in man is therefore of value, and is the necessary preliminary to surgical therapeutic procedure.

In the study of aphasia and in the localization of the various functions which take part in the use of language, clinical observation has always outrun pathological data. What is the present status of the pathology of aphasia?

It will be admitted that no doubt exists at present in regard to the pathology of motor aphasia. Facts in accord with the dictum of Broca, that a lesion of the posterior part of the third frontal convolution on the left side in right-handed, and on the right side in left-handed, persons produces a loss of the power of using language without any disturbance in the power of understanding words, are too well known and too numerous to require more than a simple statement. And the additional fact that the same effect, though usually but temporary, may follow destruction of the motor speech tract from Broca's centre to the motor nuclei of the pons and medulla rests upon positive data.² In the latter case correlated symptoms may enable the position of the lesion to be recognized during life, these symptoms being such as are usually produced by foci of disease in the internal capsule, crus cerebri, or pons varolii.

¹ Published in full in *Brain*, part xlv.

² Raymond et Artaud, *Arch. de Neurol.*, 1883, No. 20.

In regard to the pathology of sensory aphasia, the number of positive facts is by no means large. A careful search through medical literature of the past twenty years has resulted in a collection of fifty cases of aphasia of a distinctly sensory variety, which may be utilized for conclusions, and which have been tabulated. These cases will bear rigid inspection, and conform to Nothnagel's requirements in being cases of limited softening, of long duration, and thoroughly examined both clinically and pathologically.

Conclusions :

(1.) In all of these cases some form of sensory aphasia was present, and in all the lesion lay in the posterior lower third of the brain. The convolutions were found affected in the following order :

First temporal,	-	-	-	-	in 38 cases.
Second "	-	-	-	-	" 27 "
Inferior parietal,	-	-	-	-	" 21 "
Angular gyrus,	-	-	-	-	" 25 "
Supra marginal gyrus,	-	-	-	-	" 12 "
Occipital lobe,	-	-	-	-	" 19 "

In seven of the cases pure word deafness was present. The patients had lost the power to understand speech when heard, though able to read, to talk and to write (Cases III., XVIII., XX., XXX., XXXIII., XXXIV., XL.).

In all of these cases the lesion was limited to the first and second temporal convolutions in their posterior two-thirds.

In eleven of the cases pure word blindness was present. The patients had lost the power to understand words when seen, though able to understand speech and to talk (Cases II., XXIII., XXXV., XXXVI., XXXVII., XXXIX., XLII., XLIII., XLIV., XLVI., XLVII.).

In two of these cases the patients were able to write or copy, but in the remainder they had lost the power or were not tested.

In these cases the lesion was not found uniformly in one location. It affected the angular gyrus in five cases, the occipital lobe in five cases, the temporal convolutions in three cases, the inferior parietal region in three cases, and the supra marginal gyrus in two cases.

By inferior parietal convolutions it is intended to indicate those gyri which lie between the supra marginal gyrus and the angular gyrus, and which are between the interparietal sulcus and the first temporal sulcus, the area lying between P 2 and P 2' in Ecker's diagram, reproduced in "Ferrier's Functions of the Brain," p. 472.

In twenty-five of the cases the power to recall words and to name objects was impaired. This occurred in some of the cases of pure word deafness and also in some of pure word blindness. In some of these cases the power to recognize the word or name of the object when suggested by another person was preserved. And the lesion in these various cases varied widely, involving any or all of the various gyri included in the sensory speech area, or the subcortical tracts beneath them.

In seven of the cases word deafness and word blindness were present together, and yet the use of language was not lost. The patients could talk (Cases I., VII., XV., XIX., XXIV., XXIX., XXXIV.).

In these cases the lesion lay in the temporal convolutions alone in two cases, and in the remainder it extended posteriorly, involving the inferior parietal, angular and occipital convolutions.

In twenty-seven of the cases word deafness and word blindness were accompanied by more or less impairment in the power to talk. The difficulty in talking in but two cases was a difficulty in the power of pronunciation, such as occurs from lesion of Broca's centre. In all others it consisted of a use of wrong words, or unintelligent phrases, a series of words whose connection was deficient. Para-

TABLE OF CASES OF SENSORY

No. OF CASE.	AUTHOR.	REFERENCES.	LESION SITUATION.	POWER OF RECALC. WORDS
I.	Bateman	On Aphasia, 1870, p. 73	T ₁ P ₂	Impaired
II.	Broadbent....	Med.-Chi. Trans., 1872, p. 162	T ₁ P ₂	Impaired
III.	Wernicke	Aphas. Sympt. Comp., 1874 (10)	T ₁ T ₂ T ₃	Impaired
IV.	Wernicke	l. c. (2)	T ₁ P ₂	Impaired
V.	Lohmeyer	Arch. f. Klin. Chir., xiii. 323	T ₁ P ₂ sm	Impaired
VI.	Troissier	Gaz. Med. de Paris, 1874, p. 25	T ₁ P ₂ O ₂ O ₃	Impaired
VII.	Kussmaul	Ziemssen's Cyclop., xiv. p. 765	T ₁ T ₂	Lost
VII.	Kussmaul	l. c., p. 763	A T ₂ —O ₂	Impaired
IX.	Gortz	Bullet. Soc. Anat., Paris, 1876, p. 81 ..	T ₁ P ₂	Impaired
X.	Sabourin	Progres Med., 1877, p. 70.....	T ₁ T ₂ P ₂ sm.....	Impaired
XI.	Bulteau	Bullet. Soc. Anat., 1877, p. 282.....	P ₂ A O _{1,2,3}	Impaired
XII.	Broadbent....	Lancet, 1878, i 312	P ₂ A sm	Impaired
XIII.	Rudel	Dissert Breslau, 1877.....	T ₁ T ₂	Impaired
XIV.	Fritsch	Wien. Med. Presse, 1880, p. 463.....	T ₁ T ₂ P ₂ A.....	Impaired
XV.	Ball & Seguin	Arch. of Med., 1881, p. 136.....	T ₁ P ₂ A sm....	Impaired
XVI.	Chauffard	Rev. de Med., 1881, p. 939.....	T ₁ T ₂ P ₂ A sm....	Impaired
XVII.	Weiss	Wien. Med. Wochensch., 1882, p. 334..	T ₁ P ₂ A sm O... ..	Impaired
XVIII.	Girando	Rev. de Med., 1882, p. 446.....	T ₁ T ₂	Good
XIX.	Claus	Irrenfreund, 1883, p. 82	T—O	Impaired
XX.	Claus	l. c., p. 88.....	T ₁ T ₂	Impaired
XXI.	d'Heilly	Gaz. Med. de Paris, 1883, p. 22.....	T ₁ P ₂ sm.....	Impaired
XXII.	Webber	Boston Med. Surg. Jour., 1883, p. 580..	P ₂ sm....	Impaired
XXIII.	Dejerine.....	Progres Med., 1880, p. 629	P ₂ sm.....	Impaired
XXIV.	Schuetz	Charite Annalen, xiii. 418	T ₁ T ₂ A O _{1,2,3}	Impaired
XXV.	Balzer	Gaz. Med. de Paris, 1884, p. 97	T ₁ T ₂ A O ₂	Impaired
XXVI.	Rosenthal	Centralbl. f. Nerv., 1884, p. 1.....	T ₁ T ₂ A sm.....	Impaired
XXVII.	Amidon	New York Med. Jour., 1885, p. 113....	T ₁ T ₂ P ₂ A O ₂	Impaired
XXVIII.	Gunther	Zeit. f. Klin. Med., 1885, p. 16.....	T ₁ T ₂ A O _{2,3}	Impaired
XXIX.	Monakow.....	Arch. f. Psych., xvi. p. 166.....	T ₁ T ₂ A O _{2,3}	Impaired
XXX.	Seppilli	Functions local, p. 208.....	T ₁ T ₂ T ₃	Impaired
XXXI.	Seppilli	l. c., p. 205.....	T ₁ T ₂ P ₂	Impaired
XXXII.	Seppilli	l. c., p. 182	T ₁ P ₂ A O ₁₋₃ ...	Impaired
XXXIII.	Petrazzani....	Revista Sperimentale, xii. p. 235.	T ₁₋₂ Bilateral ..	Impaired
XXXIV.	Eichhorst....	Corresp. Schw. Arzte, 1886, p. 696.....	T ₁	Impaired
XXXV.	Henschen	Neurol. Centralbl., 1886, p. 424 (2)	A.....	Impaired
XXXVI.	Henschen	l. c. (3)	T ₁ T ₂	Impaired
XXXVII.	Jastrowitz....	Centralbl. f. Pract. Augenh., 1877, p.254	A.....	Impaired
XXXVIII.	Perret	Clinique Medicale, p. 137	T ₁₋₃ P ₂ A O ₁₋₃	Impaired
XXXIX.	Hun	Amer. Jour. Med. Sci., 1887, p. 154....	P ₂ A.....	Impaired
XL.	Hitzig.....	Congress f. Inn. Med., 1887, p. 166.....	T ₁ T ₂	Good
XLI.	Sigaud	Progres. Med., 1887, p. 177.....	A.....	Good
XLII.	Reinhard	Arch. f. Psych., xviii, p. 244	O ₁₋₃	Impaired
XLIII.	Bernheim	Seelenblindheit, p. 180.....	O ₁₋₃	Impaired
XLIV.	Wilbrand.....	Hecht, These de Nancy, 1887.....	O ₁₋₃	Impaired
XLV.	Laquer	Neurol. Centralbl., 1880, p. 340.....	T ₁ P ₂ O ₂	Impaired
XLVI.	Macewen	Brit. Med. Journal, 1889, Aug. 11.....	A sm.....	Good
XLVII.	Freund	Arch. f. Psych., xx. 27.....	T ₁ T ₂ A O ₁₋₃	Impaired
XLVIII.	Wigglesworth	Liverpool Med.-Chi Jour., 1887, p. 215	T ₁ A sm	Lost
XLIX.	Franks	Med. Press and Circ., 1888, p. 29.....	T ₁ T ₂	Impaired
L.	Bullen	Brain, xi. p. 514	T ₁ T ₂ A O ₂	Lost

T_{1,2,3} = First, second, and third Temporal Convolutions. O_{1,2,3}, First, second, and third the angular gyrus (A.).

WITH LESIONS AND SYMPTOMS.

[illegible]

solutions. P₂ = Inferior Parietal Convolutions lying between the supra-marginal gyrus (SM.) and

phasia is therefore the usual accompaniment of sensory aphasia. In these cases the lesion was wide in extent, involving the temporal, parietal and occipital convolutions.

It was impossible to ascertain any constant pathological difference between the cases of sensory aphasia without and with paraphasia. Nor did the power to repeat words after another seem to depend upon the relative situation of the lesion, as might be supposed from Wernicke's assertion that this defect appears with paraphasia when the temporo-frontal tract is involved. For paraphasia with inability to repeat words was found in a few cases where the lesion lay too far back to affect this tract. Paraphasia therefore may be caused by lesions in very various locations.

The analysis of the pathological lesions, therefore, does not bring out as clear a differentiation of the different forms of aphasia as might be desired.

It is evident that word deafness is due to a lesion of the first and second temporal convolutions. It is evident that word blindness may be produced by lesions lying in the region of the inferior parietal lobule, or extending either anteriorly from it into the temporal region or posteriorly into the angular gyrus and occipital lobe. It is evident that these conditions are usually associated, and when occurring together are usually accompanied by paraphasia; in these cases the lesion may lie anywhere within the limits of the sensory aphasic area, which includes the inferior parietal convolutions, the two temporal convolutions and the occipital convolutions.

In the cases here brought together the power of recalling the names of objects was impaired or lost in a large number, and the lesions producing this effect are by no means uniform in position; some lying forward in the tract within the temporal region, others about its middle in the parietal region and in the angular gyrus, others far back in the occipital lobe exclusively. The loss of power to recall the name of objects seen does not therefore indicate very exactly the seat of the disease.

But there are certain additional tests which aid in a more exact localization of verbal amnesia. If the name cannot

be recalled because the memory of it is lost, that is, because the physical basis of that memory has been not merely isolated from one connection, but actually destroyed, then it is evident that no other association will suffice to reach it, and that even recognition is impossible.³ This is the condition in absolute word deafness, which, as we have seen, is due to a lesion in the posterior two-thirds of the first and second temporal convolutions. To fail to recall a word by any means, and to fail to recognize its meaning when heard, is therefore characteristic of a lesion of this area when that lesion is extensive.

But there are cases on record where the power to recall words is impaired while the power to recognize them remains. Cases II., VIII., XXII., XXXVII., XLII., XLIII., XLIV., XLVII. illustrate this condition. This must imply that the auditory memories remain and can be reached through the auditory tract, while the association fibres alone are affected by disease. Now, the lesion in all of those cases lay in the posterior portion of the sensory aphasic area, the temporal convolutions being invaded in only two cases, and in those only at the extreme posterior portion; while in all the inferior parietal convolutions and angular gyrus or occipital lobe were affected. And in all of these cases the lesion was a deep one, invading the white tracts beneath the convolutions; in five of them the lesion was wholly subcortical (Cases II., XXII., XXXVII., XLII., XLVII.).

The conclusion, therefore, appears to be warranted that while failure to recognize a word heard implies destruction of the temporal cortical area, failure to recall the name of an object seen implies destruction of the temporo-occipital association tract in the subcortical white matter.

The latter with the former implies extensive cortical and subcortical disease; the latter without the former implies

³ Attention was first called to this fact by De Watteville, *Progrès Médical*, March, 1885.

subcortical disease only, without reference to its extent.⁴

There is a third condition which cannot be passed by in this connection, viz., psychical blindness. If an object is seen but not recognized, it implies that the visual mental image of the object is either destroyed or wholly cut off from its associations. This occurs not infrequently in lesions in the occipital lobe, either unilateral or bilateral. It is very frequently accompanied by the characteristic symptom of a lesion of the occipital lobe, namely, bilateral homonymous hemianopsia.

In twelve of the cases here collected psychical blindness was present. In six of these hemianopsia was also present. In all these cases the occipital lobe was diseased ; twice with the adjacent angular gyrus. Psychical blindness is produced not only by disease in the cortex of the occipital convolutions, but also by disease in the white tracts within the lobe. It is evident, therefore, that the memory pictures of objects lie in the occipital lobe, and a serious loss of them implies a lesion in that region. If the lesion be extensive enough to involve the cuneus, or deep enough to reach the visual tract to the cuneus as it passes beneath the angular gyrus and convexity of the occipital lobe, it will produce hemianopsia. If not, actual blindness may not accompany psychical blindness. In either case it is found that when things are not recognized, they cannot be named when seen. Hence the symptom of psychical blindness may aid in locating a lesion in the visual-auditory tract, and indicates that the lesion of that tract is in its posterior portion.

We thus have three sets of symptoms which enable us to locate a lesion in the association tract between the occipital and temporal areas, viz., loss of power to recognize the name of an object when heard (word deafness) ; loss of power to recall the name of an object recognized (verbal amnesia) ; loss of power to recognize an object seen whose

⁴ An interesting clinical example of a lesion in the association tract alone is given by Hughes Bennett in the *British Medical Journal*, 1828, i. p. 339, case third.

name is understood (psychical blindness). The first implies a lesion in the temporal end of the tract. The last implies a lesion in the occipital end of the tract. The second implies a lesion between the others, probably beneath the inferior parietal lobule. And reference to the cases cited shows that the clinical distinction is supported by the pathological finding; that the psychological hypothesis has a confirmation from the facts of disease.

The principle applied to the study of lesions in the visual-auditory tract may be extended to the consideration of other tracts. If word blindness be held, as it justly may, to be merely a variety of psychical blindness, it becomes evident that a distinction must be made between recognizing printed words, recalling printed words, reading aloud, or writing. Hence varieties in the condition of word blindness are possible.

To recognize the meaning of a sign implies integrity in the perceptive process and integrity in the associative process which joins the memory of that sign to some other mental image, giving it meaning. It is the association of two mental images which lies at the basis of any process of understanding. When we see the word "bell," it has a meaning only because the image of the word is associated with that of the object, and a destruction in the process of association will impair at once the power of recognition. It is not surprising, when this is understood, to find that the lesions producing word blindness are situated in various regions. When limited in extent and strictly cortical, the lesion producing word blindness was found in five cases in the angular gyrus and in the cortex immediately anterior to this in the inferior parietal lobule (Cases XXIII., XXXV., XXXIX., XLI., XLVI.). It is here, therefore, that the visual memory pictures are thought to lie. And, in fact, in all the cases in this collection in which the lesion involved this area and in which reading was tested, there was word blindness (twenty-one cases).

The associations between the memory of words seen and the mental image are very numerous, and reach out in dif-

ferent directions. The words *thorn*, *trumpet*, *Madonna*, call up painful, auditory, and visual memories respectively, and one can readily imagine that each of their associations might be impaired without the others being affected. But in all three cases this impairment of association might manifest itself as word blindness. Granting the hypothesis, then, that a lesion of the association tracts will produce word blindness,⁵ it is evident that tracts going out in all directions from the angular gyrus as a centre might be invaded, with the result of producing the same symptom. The two chief tracts, those usually tested, will be those to the visual area and to the auditory area. To recall the appearance of an object on seeing its name, and to pronounce the name on seeing the word, are the tests applied to these tracts respectively.

Reading understandingly and reading aloud are, therefore, different processes, the former testing a tract from the angular gyrus backward to the occipital region; the latter testing a tract from the angular gyrus forward, to the temporal region. A loss of the power to read may be associated with lesions in the occipital lobe, as in Cases XXXVII., XLII., XLIII., XLIV., in which the lesion was confined to this region. It may also accompany lesions in the posterior temporal region, which encroach upon the inferior parietal lobule, as in Cases II., X., XII., XXI., XXXVI.—in all of which, though the angular gyrus was intact, the symptom was produced.

The combination of word blindness with word deafness, in temporo-parietal lesions, and the combination of word blindness with psychical blindness and hemianopsia in parieto-occipital lesions appears to be established. Berlin's⁶ condition of dyslexia, in which a patient is fatigued unduly by reading, and which he ascribes to a subcortical lesion

⁵ This hypothesis was first broached by De Watteville, *Prog. Médical*, March, 1885. Freund appears to have overlooked this fact in a recent article, *Arch. f. Psych.*, xx., Ueber optische Aphasie und Seelenblindheit.

⁶ Berlin, *Eine besondere Art von Wortblindheit*, Wiesbaden, 1887.

beneath the angular gyrus, might well be explained by an interference with the association tracts.

Another tract which may be tested in this condition is the tract from the angular gyrus to Broca's centre. It is tested by asking a patient to read aloud, a process which may be gone through even though the patient does not understand what is read, as in Cases VII. and XXIII.⁷

Whether this tract is a direct one, or is indirect, via the temporal lobe, is still undecided; and sufficient material is waiting for definite conclusions, though in one of the cases here cited, reading aloud was possible where the temporal lobe was so much injured that word deafness was present. This case would indicate that the tract is a direct one, and if so, it must pass beneath the Island of Reil from behind forward. That it starts from the angular gyrus and passes forward into the inferior parietal lobule and supramarginal convolution is evident from the fact that in all the cases here recorded in which reading aloud was impossible, these parts were invaded by disease.

The last tracts to be tested in connection with printed language are those concerned in writing. Writing spontaneously and copying appear to test the same tract.

In all cases in which these powers were both tested, they were both lost or impaired equally. In all these cases the lesion was in or very near the angular gyrus. This tract starts then from this centre. Its direction and termination are not, however, known, as there are no autopsies upon cases of pure agraphia, excepting in the case of Sigaud (Case XLI.), where the lesion was confined to the angular gyrus and the condition was one of sensory rather than of motor agraphia.

It is evident from this review of the clinical and pathological facts in sensory aphasia that the pathological data warrant a recognition of many of the numerous forms of aphasia recently described. There are aphasias of association as well as cortical aphasias. It is necessary to recognize aphasia from lesion of the visual-auditory or occipito-temporal tract (verbal or auditory amnesia); aphasia from lesions within the occipital lobe giving rise to word blindness

⁷ A similar case is reported by Hughes Bennett, l. c.

with visual amnesia ; aphasia from lesions in the temporo-parietal region giving rise to word blindness with word deafness ; as well as the simpler forms of cortical aphasia known as word deafness, word blindness, agraphia, and motor aphasia. Thus far the clinical facts rest on pathological findings. Subjective investigation of speech processes, as well as clinical observation, may warrant further distinctions not yet resting on post-mortem records. It is evident that the various possible mental processes involved in memory must be carefully tested in every case ; and that small subcortical lesions should not be overlooked.

Inasmuch as this investigation of the pathology of sensory aphasia shows the need of more careful examination of aphasics, it may be well to suggest the lines along which such an examination should be made.

It is necessary to investigate :

1. The power to recognize objects seen, heard, felt, smelt or tasted.
2. The power to recall the names of such objects.
3. The power to recognize the names of such objects when heard.
4. The power to call to mind the objects when named.
5. The power to understand speech.

This examination will test the various sensory areas, and especially the temporal convolutions and the association tracts between these convolutions and the different sensory areas. It is also necessary to investigate :

6. The power to understand printed or written words.
7. The power to read aloud and to understand what is read.
8. The power to recall objects whose names are seen.
9. The power to write spontaneously, and to write the names of objects seen, heard, etc.
10. The power to copy and write at dictation.
11. The power to read understandingly what has been written.

These tests will determine the condition of the visual word memories in the angular gyrus, and of the connections between this area and surrounding sensory and motor areas.

It is also necessary to find out whether :

12. The power to speak voluntarily is preserved, and if not, the character of its defects.

13. The power of repeating words after another should also be tested.

DISCUSSION ON DR. STARR'S PAPER.

Dr. LLOYD had now a case of simple homonymous hemianopsia without psychical or word-blindness, and wished to know whether there was any diagnostic importance in this condition as to localization. Was there a preponderance of one hemisphere over another as regarded sensory memories of language?

Dr. STARR said that the matter of psychical blindness had recently been discussed by Wernicke and another. A lesion affecting bilaterally both occipital lobes or the association tract produces psychical blindness, but a lesion along either tract itself does not cause this condition. Nine cases of psychical blindness from lesion of one hemisphere alone had been reported.

AMERICAN ACADEMY OF MEDICINE.

The American Academy of Medicine is endeavoring to make as complete a list as possible of the Alumni of Literary Colleges, in the United States and Canada, who have received the degree of M.D. All recipients of both degrees, literary and medical, are requested to forward their names, at once, to Dr. R. J. Dunglison, Secretary, 814 N. 16th Street, Philadelphia.

**Which is the Most Powerful ?
and the Most Reliable ?
of All Pepsins ■**

Merck's Pepsin 1:2000 !

— SCALE OR POWDER —

SEE "MERCK'S INDEX," PAGES 106 AND 167

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

FEVER.

By ISAAC OTT, M.D.

BY the term fever in this paper I mean a disorder or derangement of the temperature of the body. This will include the experimental fever with high temperature and increased thermogenesis, the exceptional cases where we have high temperature with diminished thermogenesis, and the experiments where we have sub-normal temperature and a thermogenesis increased equal to a similar period on the food-day, as in Exp. 4.

Senator first studied thoroughly experimental fever with the calorimeter. He concluded that in the first stage of fever the discharge of heat is rather diminished than increased, so that at this period it is probable there is an abnormal retention. As the febrile process progressed towards its height, fluctuations exhibited themselves in the quantity of heat liberated which resembled those observed in the discharge of carbonic acid and aqueous vapor, but it could not be determined whether the amount of heat given off during the whole course of the fever was greater or less than the quantity of heat given off during the same period of apyrexia. Dr. Burdon Sanderson, in a commentary on this statement, remarks that in judging of the significance of the fact last stated it must be borne in mind that the normal with which the febrile thermogenesis is here compared is that of inanition. In the dog, when on adequate diet, the production of heat is at least fifty per cent. more

active. If therefore we were to take the animal in the ordinary condition of nourishment as our standard of comparison, we should find the heat production in fever very considerably diminished. Senator's experiments upon dogs were made as follows: each animal was placed for a sufficient time on horseflesh, which was increased or diminished until the body weight and daily discharge of nitrogen in urea and the "insensible" loss became severally constant. The diet was then continued as before until nitrogen equilibrium was once more established. This having been accomplished, fever was induced by the subcutaneous injection of perfectly fresh pus, and determinations of the same kind as had been previously made were repeated under exactly similar conditions as regards nutrition during a second period of forty-eight hours. Each experiment therefore comprised forty-eight hour periods of observation, separated from each other by an interval of several days, during which periods the production of heat, the changes of bodily weight, the daily quantities of urine and urea excreted, and respiratory and cutaneous discharges of carbonic acid and water, were determined. The carbonic acid determinations and the calorimetric observations, however, only related to limited periods of measurement each lasting an hour, repeated once or twice during the day. In only one series of calorimetric observations was the heat production measured for four hours, of which three were consecutive and the fourth separated by a four hours' interval. The calorimetric observations should have been made more frequently during the day to be of marked value.

Prof. Wood has made a number of experiments, running over about three-fourths of a day for four consecutive days. He mainly used dogs, and usually produced fever by the injection of putrid blood into the veins. The blood was injected at the end of the second day, which was the first hunger day, and he studied the fever for two consecutive days. In the pyæmic fever of dogs the H. P. was usually in excess of H. P. of fasting dogs, but less than that which could have been produced by high feeding. Usually the production of animal heat rose in the febrile state with the

temperature and with the stage of fever, but sometimes H. P. became very excessive, although the temperature of the body remained near the normal limit. In rabbits with pyæmic fever, H. P. seemed to be even greater than in health. His experiments were well calculated to determine the increase of H. P. in fever, but would have been more accurate if exactly similar periods of observations were compared on normal and fever days, on account of the hourly changes in H. P. due to rhythm.

Finkler¹ has made a most exhaustive study of experimental fever. He measured the oxygen consumed and the carbonic acid given off, and compared these with a time-unit and the weight of the animal. From his experiments the law is deduced that the consumption of oxygen is greater during the fever elevation of temperature than takes place in animals not feverish under like conditions of food and surrounding temperature. He also thinks that in fever there is increase of carbonic acid given off due to increased production. When the oxidation passes through the three phases of increase, continuance at a height and decrease, it corresponds only in a general way to elevation, continuance at a height, and decrease of temperature. Finkler arrived at the conclusion that fever is a neurosis, mainly a disease of the nervous system regulating the temperature. Pflüger's theory to explain the regulation of heat is as follows: An automatic centre which presides over the production of heat and another centre which acts upon the automatic centre as an inhibitory apparatus, and on its side stands in connection with the temperature nerves of the skin and is set into activity through the action of heat, so that coldness in general is not an irritant. When the excitation of the inhibitory centre slackens, then the automatic centre enters into activity, so that coldness of the skin corresponds to lessened formation of heat. Finkler explains the action of these centres in fever as follows: Intense increased oxidation destroys the substance generating fever. The chilly feeling and contraction of the capillaries

¹ Pflüger's Archiv., 1882.

denotes increased heat production, that in the first stage of fever a stronger excitation of the automatic centre takes place, because the nerves of the inhibitory centre are in a more or less paralytic state. In the second stage of fever, when the temperature of fever is constant, the relations of the two centres are changed. The production of heat remains as in previous stage, increased to about the same extent. In the decrease of the fever oxidation sinks below normal.

A rhythm of H.P. and H.D. exists in fever, hence all calorimetric observations should be made at the same time of day on successive days; the length of time the animal remains in the calorimeter each day should be the same; the calorimeter in observations on successive days should be nearly at the same temperature and the external temperature should be about a degree above that of the calorimeter. To produce experimental fever there are several agents which may be used. Thus solutions of hæmoglobin, albumose, peptone, fibrin-ferment, neurine, sour milk, papayotin, sulphate of ammonium and large quantities of water when injected cause experimental fever. I selected putrid blood, as it gives a fever lasting for a considerable period. My experiments were made upon rabbits and cats. In one series hourly observations were taken every six hours during the day and night, on the four successive days, the first day food being withheld as on the successive days. At the end of the second day putrid blood was injected subcutaneously or by the jugular vein, or in both ways. In another series observations were made for the first three hours, then at the sixth, eighth and twenty-fourth hour at similar periods of the successive days. I did not permit the animal to remain in the calorimeter over three hours at longest, as I feared the condition of the animal would become abnormal, although it is true the abnormality would probably be the same in each period of successive days. The calorimeter used was d'Arsonval's, and with each degree of temperature the air is above that of the calorimeter, the error was .025 F. The air was aspirated through the hollow coil, lying in the water chamber by

means of Voit's respiration apparatus, and the amount drawn through the calorimeter given in litres. The amount of heat given to or taken from the air was not calculated, as it is small and would not have changed the result. Any one who wishes to can make the calculations, as the data are given, and satisfy himself on that point. In Exp. I., the normal average temperature on the second day, or hunger day, was 102° F., and on the first fever day the average temperature was 104° F., and H. P. was decreased 7.75 thermal units; on second fever day the average temperature was 103.7 , and the decrease of H. P. was 5.0 thermal units. H. D. was decreased on the first fever day 8.0 units, and on second day 6.0 units. There was on second fever day in the morning period an increase of 2.0 units of H. P. over the same period of hunger day.

In Exp. 2, the average normal temperature on hunger day was 102.3° D., and on first fever day the temperature was 103.8° , with an increase of H. P. of 1.5 units and of H. D. of 1.3 units. On the second fever day the average temperature was 103.6° , and H. P. was decreased 2.5 and H. D. 2.7 units.

In Exp. 3, on hunger day the average temperature was 102.4° , and on first fever day 101.7 , H. P. was decreased 4.2 and H. D. 6.8 units. On second fever day the temperature was 97.5° F., and H. P. was decreased 8.2 and H. D. 6.5 units.

In Exp. 4 the temperature on hunger day was 101.4 , and on first fever day 101.4 , with an increase of H. P. 1.2 and of H. D. 2.0 units.

On second fever day the temperature was 99° , with a decrease of H. P. 0.5 and H. D. .03 units.

In Exp. 5, the average temperature on hunger day was 101.7° , and on first fever day a temperature 102.8 , with an increase of H. P. 1.0 and of H. D. a decrease of 5.0 units.

In Exp. 7, the normal temperature was 100.6 , and on first fever day the temperature was 100.9 , whilst H. P. was decreased 4. and H. D. 1.0 units.

In Exp. 8, the normal temperature was 100.7 , and on first fever day a temperature of 102.2 , with an increase of H. P. 1.2 and of H. D. 2.1 units.

On second fever day an average temperature 102.8°, with a decrease of 4.2 of H. P. and 4.0 of H. D. in units.

In Exp. 9, on hunger day an average temperature of 100.5 and on first fever day a temperature 102.6, with an increase of H. P. 5.1 and H. D. 3.0 units.

On second fever day, H. P. was increased 3.0 and H. D. decreased 3 units, whilst the average temperature was 102.7.

In the following table these experiments are summarized so that the increase or decrease of H. P. is given on the first and second fever days.

+ means increase, — decreased production.

	<i>1st fever day.</i>	<i>2d fever day.</i>
	Increase and decrease of units of H. P. com- pared with the 2d nor- mal day.	Increase and decrease of units of H. P. com- pared with the 2d nor- mal day.
Exp. 1	— 7.75	— 5.0
“ 2	+ 1.15	— 2.5
“ 3	— 4.0	— 8.2
“ 4	+ 1.2	— .50
“ 5	+ 1.0	
“ 7	— 4.0	
“ 8	+ 1.2	— 4.2
“ 9	+ 5.1	+ 2.4

By an examination of this table it is seen that five show increased heat production, whilst three indicate a decrease, on the first day.

On the second day H. P. was decreased in all except one. This is partly to be explained by food being withheld on the 1st day of the experiment.

As regards H. D. in four experiments, it was increased, and in four decreased. On second fever day it was diminished in all. At only one period of the experiments did the fever increment equal the H. P. of the first day of experiment. If, however, as in Exp. 10, on the first hunger day the H. P. and H. D. are taken for an hour, and then two drops of putrid blood are directly injected, H. P. will be increased during the next four successive hours and H. D. will also be found to be increased for the next two hours.

If Senator's experiments are taken and tabulated in a similar manner, we will have the following table. + means increased H. P., — indicates decreased H. P.

<i>Seven days.</i>	<i>1st day.</i>	<i>2d day.</i>	<i>3d day.</i>
Observation 1	— 0.86	+ .8	
" 2	— 0.28	— 1.75	
" 3	— 2.73	— 2.65	0
" 4	+ .92	+ 1.62	— .81
" 5	— .34	+ 2.55	
" 6	+ 1.28	— .89	
" 7	{ A.M. — .06 P.M. + 1.34	{ A.M. — .99 P.M. + 2.44	{ A.M. — .94 P.M. — 1.49

A glance at the table will show that it would be impossible to draw any conclusions from them, either as to increment or decrement of H. P., a conclusion to which he also came. However, they seemed to show that primarily in fever H. P. is lessened. His results are due to using blood subcutaneously, which gave him a slight fever, and not continuing his observations long enough.

If Prof. Woods' results are tabulated in a similar way, they will be found as follows :

+ means increased H. P., — decreased H. P., compared with the second day.

	<i>H. P. 1st fever day.</i>	<i>H. P. 2d fever day.</i>
Exp. 110	+ 26	+ 31
" 111	+ 2	+ 5
" 112	— 6	+ 8
" 113	— 3	+ 18
" 114		+ 37
" 116	+ 3	

An examination of Prof. Wood's results show on the first fever day an increase of H. P. in three experiments, and a decrease of H. P. in two. On second fever day there is an increase in five.

These increments are much greater than those found by me, and are partly due to observations made at dissimilar parts of the day, without regard to the diurnal rhythm.

My animals were deprived of food twelve hours before any observation had been started. His were fed during the first day, and in a few on the second day. The amount of fever as measured by the thermometer was about the same in the experiments of Dr. Wood and in mine.

All these experiments tend to the same conclusion, that experimental fever is accompanied by an increased production of heat as a rule.

Exceptionally production is decreased.

In Fig. 1 (Exp. 9) is a delineation of the access of fever, it having been studied during the first three hours and at intervals afterward. It shows that after injection per jugular of two drops of putrid blood, the heat production rises rapidly and attains its height some hours before the fever curve attains its height. At the same time the curve of H. D. is lagging behind the curve of H. P., although following it in its upward ascent. After a while the H. P. curve falls temporarily beneath the curve of H. D. and the temperature curve falls. It will be seen normally and during the fever in the curve of H. P. that it exhibits fluctuations, a fact pointed out by Senator. The fluctuations of H. P. are greater in fever. I believe the fluctuations are due to the action of external agencies upon the thermotaxic, thermogenetic and thermolytic apparatus, which are playing at see-saw, at one time making H. P. greater than H. D., at another making H. D. greater than H. P.

In Fig. 2 (Exp. 1) there is an illustration of a high temperature, although H. P. and H. D. have fallen below normal of the hunger day or second day. In Exp. 4, we see that during three-fourths of the last fever day the temperature is below normal, and at the last observation H. P. is five units greater than those of same period on hunger day. The question arises how is Fig. 2 to be explained?

Dr. Donald McAlister has given an explanation of this. Suppose a tall vessel containing water, the level of the water representing temperature. Let two pipes be connected with this vessel, one conveying water, the other carrying it off. Let the inlet and exit tubes be each provided with a stop-cock, and let the two stop-cocks be

connected by a rigid link which insures that they always turn together and by the same amount. If to start with, the inflow and outflow are equal, then however I move the linked stop-cocks, the height of the water will be the same. Now remove the rigid link and connect the stop cocks by a spiral spring. If now you move the inflow stop-cock so as to increase the flow, the outflow one will not at once follow, and, the balance being broken, the level of water will rise. But shortly the elasticity of the spring comes into activity, the outflow is equal to the inflow and the rise will cease, but the new high level will be maintained. Every movement of either stop-cock will affect the level, which will fluctuate accordingly, but its height at any moment will not be an index of the amount of inflow at that moment, The inflow may be slight while the level is high. If now you substitute H. P. for inflow and H. D. for outflow, and the rigid link will represent the healthy thermotaxic mechanism, then when this is weakened or relaxed or broken the steadiness of the normal level is impossible.

Fig. 2 amply sustains this explanation. I have tried to determine what part of the thermal apparatus is the most essential for the development of fever. The skull of rabbits, under ether, has been trephined and part of the vault broken away, then all the cortex accessible destroyed with a blunt probe without disturbing the basal ganglia. After the animal recovered from the ether and shock, putrid blood was injected by the jugular, and still fever ensued. Recently Dr. Sawadowski has published a note proving that after removal of the corpora striata in dogs, putrid blood per jugular could not cause fever. He also demonstrated that antipyrin exerted its action through the corpora striata, for upon their removal no antipyretic effect was noticed. I have made a series of experiments upon this subject, and generally noted that after extirpation of the anterior ends of the corpora striata and the injection of putrid blood, a rise of temperature. If the striate bodies are completely extirpated, there is usually no rise by putrid blood. However, I have been fortunate enough in one experiment (14) to obtain a rise of half a degree, after removal of the striate

bodies, by injections of putrid blood. Notwithstanding the shock by removal of these bodies, the other basal centres will still respond to the septic poison. This rise is not due to simple extirpation of corpus striatum, for after the operation the temperature falls in rabbits for several hours.

It now remains to consider how far these facts are supported by clinical experience. Liebermeister sought to determine the H. D. in man by placing fevered persons in cold baths and noting the amount of heat given to the water. He calculated the amount of cooling which the water would undergo without the patient during the same time the patient was in it. This method is liable to many errors, which he sought to overcome in part. The loss of heat to the air and from the lungs could not be noted. He arrived at the conclusion that when baths of the same temperature are employed "without exception the loss of heat in the fever patient is greater than in the well person. The recent experiments of Fredericque and Quinquad prove that the cold bath itself increases H. P. and necessarily H. D. Prof. Leyden attempted the solution of the problem in a different manner upon patients affected with relapsing typhoid, and pneumonia. To determine the surface loss of heat a water calorimeter was employed, which was constructed on the same principle as the one usually employed in calorimetric work, except there was no provision for the continuous passage of a current of air. The apparatus consisted of a copper chamber, in which the limb was contained. It was two feet long and one foot wide. It was surrounded by a cylinder of zinc of corresponding form, but from three to four inches wide. The outer wall of the water chamber was protected from abstraction of heat by a thick padding of non-conducting material enclosed in a wooden case. The water was agitated by means of a special apparatus. The open end of the chamber was lined by an annular cushion of india-rubber, which, when the limb was introduced, occupied the space between its surface and that of the copper, so as to close the chamber air-tight. The leg of the patient was introduced into the apparatus, and the rubber cap covered the knee. When an observation was to be

made the calorimeter was warmed to the air temperature of the room, and each experiment lasted two hours. The leg was placed on a wooden support, so as not to touch the copper, and clothed with a blanket of the same thickness as the opposite leg. He found in a healthy person the mean rise of temperature in the apparatus, and compared his fever observations with it. He arrived at the conclusion, first that the discharge of heat is increased in fever whether the temperature is constant, falls, or rises. Consequently it is certain that the production of heat is increased. In high fever the quantity of heat given off is from half as much again as the normal to twice as much. The most rapid discharge of heat takes place in the critical stage when the temperature is rapidly sinking. It may then be twice or even three times as great as normal. This rapid critical dissipation takes place with profuse sweating.

Senator believed that Leyden's observations proved two facts: 1st, that with the exception of the initial stage, the discharge of heat is considerably increased, although by no means constantly; second, that the activity of the discharge is not proportional to the bodily temperature, for it may be less when the temperature is high than when it is lower, it may be normal when the temperature is above normal, always attaining its maximum in the stage of defervescence with critical sweating. From Leyden's data he concludes the average loss of heat in fever to be seventy to seventy-five per cent., although his calorimetric observation on animals gave no definite result.

Senator's general conclusion on fever was that the discharge of heat is in the outset of fever, during the rigor, not increased but diminished; during the height of the fever it is on the whole increased seventy-five per cent, and considerably more during the critical defervescence.

Dr. Sanderson has also calculated the amount of heat in fever by using Frankland's heat value of the immediate principles of food—albumen, fat, and carbonic hydrates—when converted by oxidation into urea and carbonic acid. He arrives at the conclusion that less heat is produced in fever than when the man is fed up to food-limit, but very

much more heat is produced in the febrile state than when the man is kept without food. It must be remembered that Liebermeister and Leyden believe H. P. in fever is increased even beyond that of the food limit, an increase absolute.

Dr. Carl Rosenthal has recently investigated fever by means of a calorimeter somewhat similar to that used by Prof. Leyden, except the constricting band of rubber is wisely omitted. He used the arm instead of the leg. He arrives at the conclusion that the elevation of temperature in fever is mainly due to a diminution of the H. D. There takes place simultaneously a heaping up of heat produced in the normal manner through the diminution of the H. D. It is absolutely unnecessary to have an accompanying increase of H. P. The diminution of H. D. happens in the following way. The fever agent circulating in the blood acts specifically upon a vaso-motor centre, either by a direct excitation of the vaso-constrictors, a vaso-motor contraction and diminution of H. D., or the vaso-dilators have their activity reduced, by which the vaso-constrictors obtain control and thus in an indirect manner diminish H. D. Whilst he believes diminished H. D. to be the principal and, in the first place, the cause of the fever, he holds it secondarily to be perhaps due to an increased production, whose origin is to be sought in an increased chemical metamorphosis which is expressed by increased discharge of urea, and is caused perhaps by changes in the blood itself by the abnormal elevation of its temperature. These experiments upon an extremity are not as satisfactory as they might be. The better plan is to do as Langlois did, put the fevered child in a calorimeter completely surrounding it, and then study H. P. and H. D. of the whole body. This is the more easy, as d'Arsonval and I have constructed a calorimeter for observations upon the whole body of a man. In a hundred and eleven observations, mainly on fever of broncho-pneumonia of children, with some on varicella, Langlois found the H. P. to be increased corresponding with the rise of temperature, but the radiation of heat is not always in constant relation with the temperature. In chronic maladies with hyperthermia, there

was a diminution of the H. P., whilst in acute disease the augmentation was ten to fifteen per cent. of H. P.

If now we take the experimental data and the majority of the clinical conclusions, the result must be arrived at that fever is usually temporarily accompanied by an increased production of heat, an increase beyond that normally seen in a fasting state, but not equal to the amount produced upon a full diet. Fever cannot be due to retention of heat in my experiments, as injections of putrid blood do not elevate the arterial tension but lower it, which would cause greater dissipation. It is probable that in man during the chill the heat dissipation is temporarily lessened, and co-operates with the increased production to elevate the temperature. I wish to state here that the temperature of fever has no relation to the increased production of heat. The temperature is decided by the relation between H. P. and H. D.; they may be high or low in amount, as my curves show. All calorimetric experiments upon pyæmic fever show that H. D. is not usually decreased but increased at the time fever is generated. The researches upon albumose, peptone and neurin fever, although not accurate, support these views. In peptone fever there is a temporary fall of H. P., but the temperature rises as it does in albumose fever, but in albumose fever the H. P. does not fall but rises immediately. It is thus possible to partly differentiate an albumose from a peptone, the peptone producing a temporary decrease of H. P. before it rises, whilst an albumose causes an immediate rise of H. P. There is no reason to believe that the physical and chemical processes of fever differ from those normally going on. The thermotaxic centres at the base of the brain neither inhibit nor excite H. P.; all they do is to maintain the balance between H. P. and H. D., so as to keep the temperature at 98.4° F. The two cortical thermotaxic centres also assist. Now, in fever, these thermotaxic centres are so disordered that it is mainly the basal thermotaxic which are affected that the relation between H. P. and H. D. is so disturbed that a higher temperature results and continues. This is the part affected in fever. Neither increased pro-

duction, diminished dissipation or even high temperature are necessary to constitute a fever, but it is only a disease of thermotaxic centres and mainly the four basal thermotaxic. A similar theory has been put forth by Liebermeister, but he did not prove it, nor did he understand the mechanism or the location of these basal thermotaxic centres in the production of the temperature part of fever.

My experimental researches lead me to believe that fever is due to an agent from within or without which deranges the harmony of the thermotaxic, thermogenetic and thermolytic apparatuses, by which in the initial stage the metabolism of the tissues is usually temporarily increased and this increment is usually greater than that generated upon a restricted amount of nutriment. It is highly probable that during the chill heat dissipation is temporarily diminished, but it usually follows the fluctuations of heat production. The four basal thermotaxic centres play the most important part in the temperature-phenomena of fever. That neither increased production nor diminished dissipation are necessary to constitute fever is shown in Fig. 2, where heat production is diminished, although the temperature is elevated, and in Exp. 4, at one period the temperature is subnormal, yet the heat production is greatly increased above that seen on a similar period of the preceding day.

High temperature does not cause gravity in fever, for in nervous disorders and in relapsing fever we have high temperatures, 106° F., and no serious symptoms are present. High temperature is an indication of danger in specific fever, not the cause of it. But temperature is only a part of a specific fever, there are many other morbid processes going on, the essence of which has not been grasped. Sir William Jenna puts the facts tersely when he states: "There can be no doubt that the necessity for a healthy condition of the blood is as essential to the formation of normal secretions as a healthy state of the nervous system. But while we think there is strong evidence in favor of the primary affection of the blood and of the wide-spread and fearfully severe influence on the system generally of the very deep lesion which in many cases we can demonstrate

the blood to have experienced independently of mere admixture of excess of excrementitious matters, we by no means exclude the nervous system or any other part of the body from a share in the production of the symptoms of fever."

It has been observed (very rarely I think) that in meningitis, peritonitis, and certain cases of typhoid fever, that the temperature is normal or subnormal. I have shown that in the cortex of lower animals are localized thermotaxic centres, the cruciate and Sylvian, whose function it is to act in harmony with the basal thermotaxic centres to regulate the temperature of the body. In a recent paper I have also shown that in man there are very good reasons to believe in the localization of cortical thermotaxic centres. Now in meningitis the inflammation of the membranes by contiguity may so disorder the thermotaxic centres of the cortex that the temperature may become subnormal, instead of being above normal by an alteration of the harmony between the heat production and heat dissipation. In lower animals it is well known that peritoneal irritation as well as in man greatly reduces the force and frequency of the heart by reflexly stimulating the carotid-inhibitory apparatus and thus keep metabolism at a low point in the primary stage of a peritonitis. In the subnormal cases of temperature of typhoid patients it is easy to see that the disorder of the thermotaxic centres may be such that the relation of H. P. to H. D. is so arranged that the temperature becomes subnormal. An antipyretic usually temporarily produces this state of affairs in normal state. That acute observer, Dr. W. Hale White, has propounded a theory that the cause of fever in certain cases acts upon the nervous centres not directly, but indirectly through the nerves. He states that the rise of temperature noted in fever which is symptomatic of local inflammation bears no relation to the extent of the inflammatory disturbance and that the tension of the neighboring tissues plays a more important part in the production of fever than does the amount of inflammation. It is certainly the case, he states, that in abscess the temperature as a rule is highest where there is pain, and pain in abscess

means tension. Dr. White believes that the "calorific centres" of the brain are affected reflexely by the tension of the inflamed parts acting as a stimulant which takes off their normal inhibition. It has not been my experience that irritation of a sensory nerve will elevate the temperature for any length of time, except in cases of poisoning by atropin, where irritation causes a rise. I shall elaborate the mechanism of the thermotaxic centres in another paper on thermo-polypnœa.

Appended are the experiments upon which the preceding statements are based.

In these experiments the first day without food is called the "food day;" the second day, the "hunger day."

- A. T. means air temperature.
 C. T. " calorimeter temperature.
 E. T. " exit tube " "
 R. T. " rectal " "
 H. D. " heat dissipation.
 H. P. " heat production.
 Weight " weight in pounds.
 Litres " litres of air.

EXP. I.—Cat.

Food Day.

P. M.	A. T.	C. T.	E. T.	R. T.	Weight 4.42
8.00	74.9	72.4	24.1	103.2	
9.00	74.3	73.5	24.	101.6	

+ 1.1

— .6

H. P. = 40.02 H. D. = 45.89

A. M.	A. T.	C. T.	E. T.	R. T.	Weight 4.40
12.30	75.0	73.4	23.9	102.9	
1.30	75.1	74.55	25.7	101.0	

+ 1.15

— 1.9

H. P. = 39.58 H. D. = 47.97

A. M.	A. T.	C. T.	E. T.	R. T.	Weight 4.34
8.20	73.3	72.95	24.3	102.9	
9.20	73.8	73.9	24.4	102.0	

+ .95

— .9

H. P. = 36.39 H. D. = 39.63

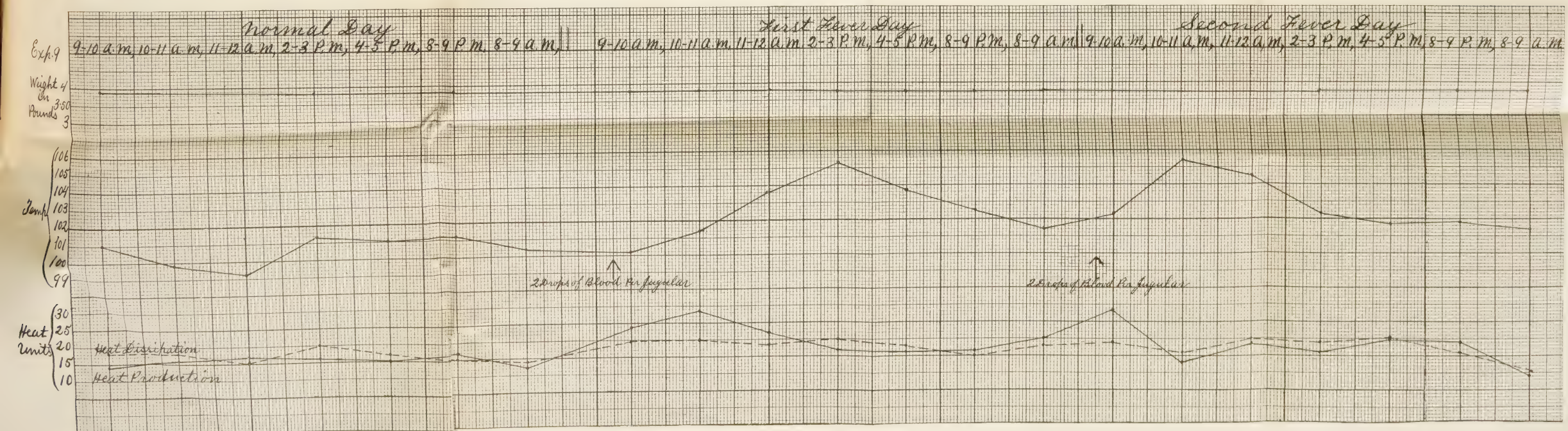
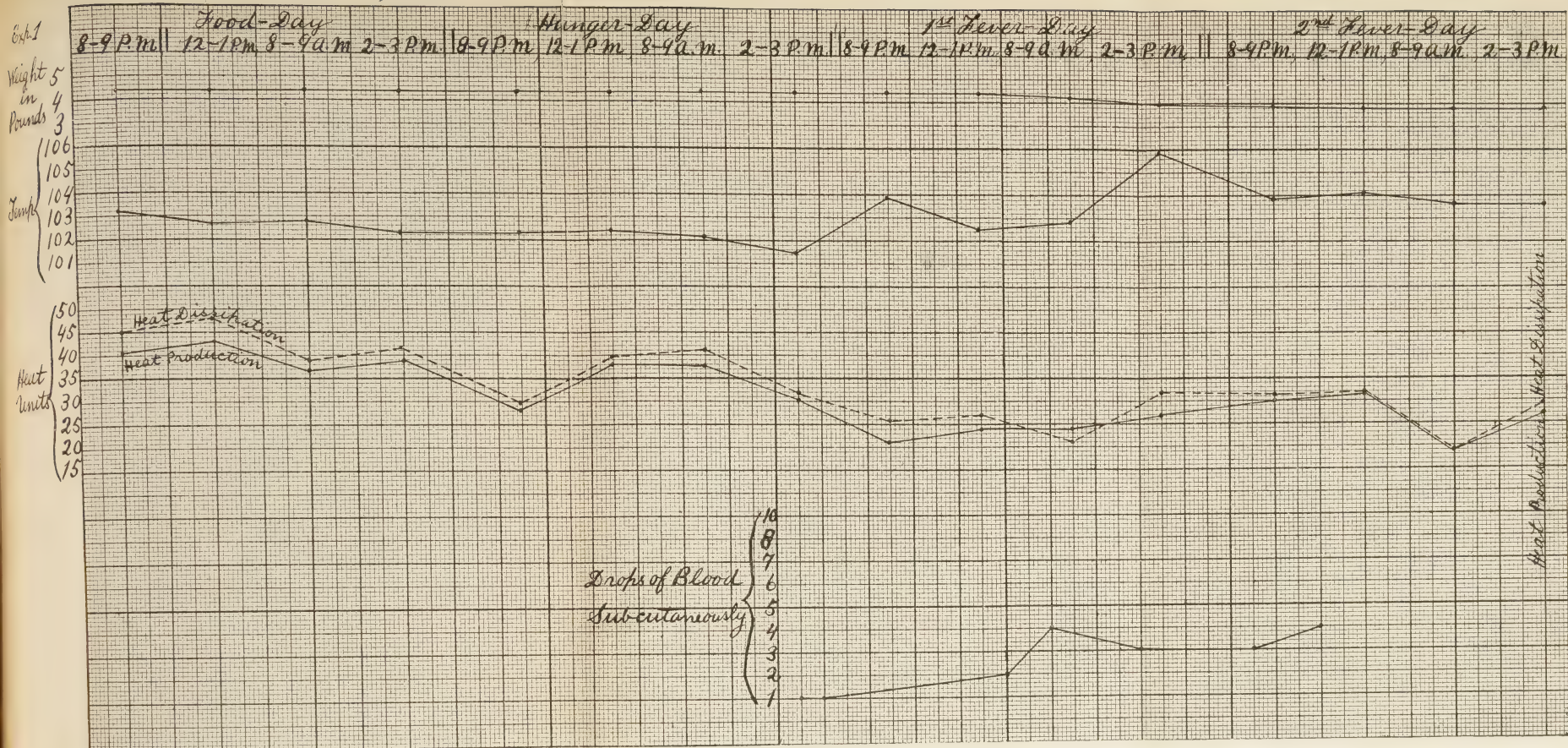
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P. M.				
2.00	75.0	73.6	23.9	102.2
3.00	74.6	74.6	24.5	101.3

Weight 4.34

+ 1. — 1.

H. P. = 38.12 H. D. = 41.72

Hunger Day.

P. M.	A. T.	C. T.	E. T.	R. T.
8.10	75.8	74.7	25.0	102.4
9.10	75.4	75.4	24.5	102.2

Weight 4.34

+ .7 — .2

H. P. = 23.49 H. D. = 29.20

A. M.				
12.30	76.1	75.55	26.3	102.4
1.30	78.1	76.50	26.2	102.0

Weight 4.28

+ .95 — .4

H. P. = 38.21 H. D. = 49.63

A. M.				
8.15	76.4	74.95	26.0	102.0
9.15	77.5	75.95	25.9	101.4

Weight 4.12

+ 1.00 — .6

H. P. = 39.67 H. D. = 41.72

P. M.				
1.45	79.3	76.0	26.5	101.5
2.45	78.7	76.75	26.2	101.4

Weight 4.12

.75 — .4

H. P. = 30.95 H. D. = 31.29

P. M.				
3.00	1 gtt. of blood several months old, subcutaneously.			
4.00				102.8
5.15				103.1

First Fever Day.

P. M.	A. T.	C. T.	E. T.	R. T.
7.55	80.6	78.1	26.7	103.9
8.55	79.3	78.75	26.6	102.3

+ .65 — 1.6

H. P. = 21.27 H. D. = 27.11

A. M.				
12.42	78.7	78.65	25.8	102.5
1.42	79.5	79.3	27.0	101.8

Weight 4.2

.7

H. P. = 24.77 H. D. = 27.11

A. M.					
1.43	2	gtts. of blood several months old, subcutaneously.			
6	3	"	"	"	"
8	3	"	"	"	"
8.39	1	"	"	"	"
8.40		75.6	75.65	25.6	103.8
9.40		76.2	76.2	25.9	104.3
					<hr/>
					.5
		H. P. = 24.60 H. D. = 22.94			

10.00	3	gtts. of blood several months old, subcutaneously.			
11.20					104.4
P. M.					
1.20	3	gtts. of blood several months old, subcutaneously.			
					Weight 3.98
2.05		78.7	76.1	26.4	105.8
3.05		79.5	76.9	26.7	104.2
					<hr/>
					16.
		H. P. = 27.60 H. D. = 33.37			

Second Fever Day.

P. M.					
8.14	3	gtts. of blood several months old, subcutaneously.			
		A. T.	C. T.	E. T.	R. T. Weight 3.98
8.15		81.3	77.95	27.2	103.9
9.15		78.7	78.7	26.3	103.65
					<hr/>
			+.75		-.25
		H. P. = 30.46 H. D. = 31.28			

A. M.					
12.45	4	gtts. of blood several months old, subcutaneously.			
					Weight 3.96
12.47		78.6	78.6	27.2	104.0
1.47		80.3	79.5	28.2	104.1
					<hr/>
			+.9		.1
		H. P. = 37.87 H. D. = 37.54			
8.04	10	gtts. of blood several months old, subcutaneously.			
					Weight 3.88
8.05		77.1	77.0	25.4	103.6
9.05		76.4	77.5	25.5	103.6
					<hr/>
			+.5		
		H. P. = 20.86 H. D. = 20.86			

11.50 104.0
P. M.

1.54 10gtts. of blood several months old, subcutaneously.

1.55 77.8 77.1 26. 103.6

2.55 78.8 77.8 26.6 103.5

+ .7 — .1

H. P. = 28.88 H. D. = 29.20

3 15 1 gtt. of blood several months old, per jugular.

3.20 79.1 77.9 26.7 104.2

4.20 80.1 78.45 27.6 105.3

.55

H. P. = 26.48 H. D. = 22.94

EXP. 2.—Rabbit.

Food Day.

A. M. A. T. C. T. E. T. R. T. Weight 5.16

8.10 69.7 68.85 22.5 102.4

9.10 71.1 69.9 22.5 101.5

+ 1.05 — .9

H. P. = 40.05 H. D. = 43.80

P. M. Weight 5.16

1.45 71.6 69.8 22.5 102.0

2.45 91.8 70.95 22.1 101.4

+ 1.15 — .6

H. P. = 45.41 H. D. = 47.97

Weight 5.1

7.35 71.9 71.1 22.2 102.9

8.35 73.5 72.05 22.6 102.4

+ .95 — .5

H. P. = 37.52 H. D. = 39.63

Weight 4.96

A. M. 12.42 72.5 72.2 22.2 102.4

1.42 75.3 73.2 22.6 102.7

+ 1.0 + .3

H. P. = 42.72 H. D. = 41.72

Hunger Day.

A. M. A. T. C. T. E. T. R. T. Weight 4.94

8.35 72.8 72.3 27.5 102.3

9.35 74.6 73.2 27.4 102.4

+ .9 + .1

H. P. = 37.95 H. D. = 37.54

P. M.				Weight 4.80
1.45	76.4	73.35	102.2	
2.45	77.0	74.2	102.4	

.85 + .2

H. P. = 36.72 H. D. = 35.46

Weight 4.80

8.02	78.3	75.55	102.7
9.02	78.0	76.55	102.8

+1.00 + .1

H. P. = 40.02 H. D. = 39.63

Weight 4.74

A. M.			
1.58	78.	76.75	102.3
2.58	78.1	77.49	102.8

+ .74 + .5

H. P. = 32.84 H. D. = 30.87

3.7 1 gtt. of blood several months old, subcutaneously.

First Fever Day.

A. M.				Weight 4.68
8.14	1 gtt. of blood several months old,			subcutaneously.
8.15	77.1	76.85	103.4	
9.15	77.9	77.8	103.7	

.95 + .3

H. P. = 40.83 H. D. = 39.63

P. M.				Weight 4.6
1.32	1 gtt. of blood several months old,			subcutaneously.
1.33	80.1	77.95	103.9	
	79.7	78.85	103.7	

.90 — .2

H. P. = 36.78 H. D. = 37.54

7.48	3 gtt. of blood several months old,			subcutaneously.
7.50	81.0	79.5	103.8	
	78.3	80.3	103.9	

+ .8 + .1

H. P. = 33.74 H. D. = 33.37

A. M.			
12.30	5 gtt. of blood several months old,		
12.31	80.4	80.3	105.1
1.31	81.5	81.2	104.4

+ .9 + 1.3

H. P. = 42.53 H. D. = 37.54

Second Fever Day.

A. M. 5 gtts. of blood, subcutaneously. Weight 4.40

8.6	79.6	79.6	103.6
8.7			
9.7	82.2	80.35	103.8
		<hr/>	<hr/>
		.75	+ .2

H. P. = 32.02 H. D. = 31.29

1.56 12 gtts. of blood, subcutaneously. Weight 4.30

P. M.			
1.58	83.1	80.10	103.6
2.58	81.1	80.9	103.8
		<hr/>	<hr/>
		+ .8	+ .2

H. P. = 34.08 H. D. = 33.37

8.0 10 gtts. of blood injected into the peritoneal cavity.

8.5	80.1	80.2	103.8
9.5	81.6	81.0	105.2
		<hr/>	<hr/>
		+ .8	+ 1.4

H. P. = 38.29 H. D. = 33.37

A. M. 1. 15 gtts. of blood into peritoneal cavity. Weight 4.14

1.2	81.3	81.0	103.6
2.2	82.8	81.8	103.1
		<hr/>	<hr/>
		+ .8	— .5

H. P. = 31.66 H. D. = 33.37

EXP. 3.—Dark-colored rabbit.

Food Day.

P. M. A. T. C. T. E. T. R. T. Weight 3.74
12.35 74.6 74.3 24.6 102.3 Litres of air
drawn through
calorimeter

1.35 75.1 75.1 24.0 101.9 124.25

H. P. = 31.51 H. D. = 33.37

8.07 74.1 73.85 24.4 102.4 Weight 3.72
Litres of air
126.62

9.07 75.4 74.5 24.6 101.8

+ .4 — .6
H. P. = 25.27 H. D. = 27.12

A. M.					Weight 3.66
2.00	75.9	75.15	24.7	102.1	Litres 111.50
3.00	77.3	75.9	25.0	101.2	

H. P. = 31.59 H. D. = 31.29

7.55	77.8	76.1	26.3	102.8	Weight 3.56
8.55	78.3	76.8	26.0	101.5	Litres 123.67

— 1.3

H. P. = 25.36 H. D. = 29.20

Hunger Day.

P. M.					Weight 3.54
12.37	77.3	77.3	25.9	102.1	Liters 118.37
1.37	78.1	77.8	26.2	101.8	

+ .5

— .3

H. P. = 19.98 H. D. = 20.86

8.17	72.0	71.7	23.6	101.8	Weight 3.5
9.17	73.2	72.35	22.8	100.7	Litres 116.11

+ .65

— 1.1

H. P. = 23.92 H. D. = 27.11

A. M.					Weight 3.44
1.45	73.3	72.4	24.3	101.5	Litres 113.75
2.45	74.3	73.0	24.2	101.2	

+ .6

— .3

H. P. = 24.17 H. D. = 25.03

7.55	74.0	73.1	24.6	101.5	Weight 3.36
8.55	75.6	73.8	24.3	100.7	Litres 106.12

+ .7

— .8

9.00 1 gtt. of blood several months old, subcutaneously.

First Fever Day.

P. M.					Weight 3.32
12.35	1 gtt. of old blood, subcutaneously.				Litres 106.05
12.36	74.2	74.18	24.1	101.0	
1.36	75.7	74.80	25.1	100.8	

+ .62

— .2

H. P. = 25.31 H. D. = 25.86

8.00	5 gtt. of blood, subcutaneously.				Weight 3.24
8.20	67.6	68.7	21.4	100.9	Litres 47.57
9.20	70.0	69.2	21.4	99.6	
		<u>+ .5</u>		<u>— 1.3</u>	
	H. P. = 17.31		H. D. = 20.86		
9.30	5 gtt. of blood 13 days old, subcutaneously.				
11.20	5 gtt. of blood 13 days old, into peritoneal cavity.				
12 M.				99.6	
A. M.					Weight 3.18
1.45	5 gtt. of blood 13 days old, subcutaneously.				
1.50	72.1	69.4	23.0	95.6	Litres 211.00
2.50	74.1	69.98	22.9	94.8	
		<u>+ .58</u>		<u>— .8</u>	
	H. P. = 22.08		H. D. = 24.19		
					Weight 3.14
7.45	71.5	70.6	22.5	90.6	Litres 113.35
8.45	71.2	71.2	22.4	88.2	
		<u>+ .6</u>		<u>— 2.4</u>	
	H. P. = 18.78		H. D. = 25.03		
9.30	Animal dead.				

EXP. 4.—White rabbit.

Food Day.

P. M.	A. T.	C. T.	E. T.	R. T.	Weight 3.58
1.44	75.1	75.1	24.0	100.9	Litres 115.37
2.44	76.8	75.7	24.9	101.7	
		<u>+.6</u>		<u>+ .8</u>	
	H. P. = 27.40		H. D. = 25.03		
					Weight 3.5
9.20	77.0	74.55	24.8	101.3	Litres 119.87
10.20	76.2	75.15	24.7	100.9	
				<u>- 4</u>	
	H. P. = 23.87		H. D. = 25.03		
					Weight 3.52
A. M.					Litres 129.70
3.00	77.3	75.9	25.0	101.0	
4.00	77.3	76.35	25.3	101.1	
		<u>+.45</u>		<u>+ .1</u>	
	H. P. = 19.06		H. D. = 18.77		

8.55	78.3	76.8	26.0	101.8	Weight 3.50
9.55	78.5	77.35	26.3	102.0	Litres 119.82
H. P. = 23.52 H. D. = 22.94					

Hunger Day.

P. M.					Weight 3.42
1.37	78.1	77.8	26.2	102.1	Litres 118.44
2.37	82.8	78.35	28.2	102.2	
				+.55	+.1
H. P. = 23.22 H. D. = 22.94					

9.17	73.2	72.35	22.8	101.4	Weight 3.34
10.17	73.0	72.8	22.8	101.0	Litres 113.55
				+.45	— .4
H. P. = 17.66 H. D. = 18.77					

A. M.					Weight 3.28
2.45	74.3	73.0	24.2	101.4	Litres 102.41
3.45	74.8	73.6	24.0	100.3	
				+.6	— 1.1
H. P. = 22.03 H. D. = 25.03					

8.55	75.6	73.8	24.3	100.8	Weight 3.28
9.55	75.5	74.3	24.3	100.7	Litres 97.38
H. P. = 20.59 H. D. = 20.86					

10.05	1 gtt.	of blood several months old,	hypodermically.		
1.32	2 gtt.	"	"	"	"

First Fever Day.

P. M.					Weight 3.22
1.37	75.7	74.8	25.1	102.3	Litres 169.12
2.37	78.9	75.35	26.1	102.3	
				.55	— .0
H. P. = 22.94 H. D. = 22.94					

9.15	5 gtt.	of blood several months old,	subcutaneously.		Weight 3.14
9.20	70.0	69.2	21.4	100.2	Litres 94.47
10.20	71.7	69.7	21.9	100.7	
H. P. = 22.16 H. D. = 20.86					

A. M.					Weight 3.10
2.45	5 gtt. of blood 13 days old, subcutaneously.				
2.50	74.1	69.98	22.9	101.3	Litres 106.75
3.50	72.5	70.60	22.2	100.2	
		<hr/>		<hr/>	
		+ .62		+ 1.1	
	H. P. = 23.02		H. D. = 25.86		

					Weight 3.10
8.44	10 gtt. of blood, 13 days old.				
8.45	71.7	71.2	22.4	102.1	Litres 103.82
9.45	74.0	71.8	23.7	100.2	
		<hr/>		<hr/>	
		+ .6		— 1.9	
	H. P. = 20.14		H. D. = 25.86		

Second Fever Day.

P. M.					
1.35	5 gtt. of blood 13 days old, subcutaneously.				
1.37	71.7	71.65	23.2	101.7	Litres 103.70
2.37	73.0	72.20	22.8	100.3	
		<hr/>		<hr/>	
		.55		— 1.4	
	H. P. = 19.34		H. D. = 22.94		

A. M.					Weight 3.00
8.00	1 gtt. of blood 13 days old, per jugular.				
9.10	69.5	69.7	21.8	99.1	Litres 73.02
10.10	72.2	70.05	22.7	100.4	
		<hr/>		<hr/>	
		+ .35		+ 1.3	
	H. P. = 17.83		H. D. = 14.60		

2.35	5 gtt. of blood 13 days old, subcutaneously.				
2.40	71.3	69.7	22.5	98.4	Litres 62.62
3.40	73.2	70.25	22.6	97.5	
		<hr/>		<hr/>	
		+ .55		— .9	
	H. P. = 20.75		H. D. = 22.94		

8.50	73.1	71.0	22.8	97.1	Litres 105.75
9.50	74.3	71.6	23.2	97.2	
		<hr/>		<hr/>	
		+ .6		+ .1	
	H. P. = 25.27		H. D. = 25.03		

EXP. 5.—Fawn-colored rabbit.

Food Day.

A. M.	A. T.	C. T.	E. T.	R. T.	Weight 3.5
8.10	67.4	67.3	21.1	101.9	Litres 55.52
9.10	69.8	68.05	21.1	101.8	
H. P. = 31.00		H. D. = 31.29.		Weight 3.46	

P. M.					Litres 33.32
1.45	73.1	68.95	22.6	102.5	
2.45	74.9	69.85	24.3	101.8	
		.9		— .7	
H. P. = 35.53		H. D. = 37.54			

P. M.					Weight 3.34
8.15	76.6	72.55	2.40	102.1	Litres 73.85
9.15	76.1	73.25	2.40	101.8	
		+ .7		— .3	
H. P. = 28.37		H. D. = 29.20			

A. M.					Weight 3.34
12.45	73.7	74.0	23.6	102.1	Litres 120.
1.45	77.	74.65	24.7	102.1	
		+ .65		.0	
H. P. = 27.11		H. D. = 27.11			

Hunger Day.

A. M.					Weight 3.3
8.15	73.3	74.25	24.1	101.7	Litres 105.0
9.15	75.9	74.7	24.5	101.7	
		+ .45		.0	
H. P. = 18.77		H. D. = 18.77			

P. M.					Weight 3.3
1.45	77.2	75.3	25.6	101.4	Litres 115.0
2.45	78.0	75.8	25.5	102.0	
		+ .5		+ .6	
H. P. = 22.50		H. D. = 20.86			

					Weight 3.28
7.55	78.2	76.3	26.3	101.9	Litres 117.5
8.55	78.6	76.85	26.0	102.0	
		+ .55		+ .1	
H. P. = 23.21		H. D. = 22.94			

A. M.					Weight 3.28
12.40	77.6	77.45	26.7	102.0	Litres 115.0
1.40	79.7	78.0	25.7	101.8	

+ .55 — 2.

H. P. = 22.4 H. D. = 22.94

2.55 1 gtt. of blood six days old, subcutaneously.
 7.00 3 gtt. " " " " "

First Fever Day.

A. M.					Weight 3.08
8.20	73.5	73.4	24.3	102.0	Litres 100.0
9.20	75.3	73.8	24.4	102.3	

+ .4

H. P. = 17.44 H. D. = 16.68

8.30 3 gtt. of blood six days old, subcutaneously.
 12.00 5 gtt. " " " " "

P. M.					Weight 3.0
1.50	76.	74.4	25.2	103.6	Litres 72.5
2.50	76.9	75.0	24.9	103.6	

+ .6 .0

H. P. = 25.03 H. D. = 25.03

5.30 7 gtt. of blood six days old, hypodermically.
 6.30 Lying on side, rolling from side to side, clonic and
 tonic convulsions, opisthotomos, trismus, death.

EXP. 6.—White rabbit.

Food Day.

A. M.	A. T.	C. T.	E. T.	R. T.	Weight 2.9
9.10	69.8	68.05	21.1	101.1	
10.10	69.9	68.7	21.1	101.6	Litres 140.30

+ .65 .5

H. P. = 28.31 H. D. = 27.11

P. M.					Weight 2.82
2.40	Animal aborted.				
2.45	74.9	69.85	24.3	101.4	Litres 93.10
3.45	77.	70.6	24.1	101.8	

+ .75 + .4

H. P. = 30.36 H. D. = 31.29

9.15	76.1	73.25	24.	101.5	Weight 2.76
10.15	76.6	73.9	24.8	101.6	Litres 106.35

+ .65

+ .1

H. P. = 27.34

H. D. = 27.11

A. M.

1.45 77. 74.65

24.7 101.5

Weight 2.72
Litres 125.00

2.45 75.7 75.3

24.7 101.7

+ .65

+ .2

H. P. = 27.51

H. D. = 27.11

Hunger Day.

A. M.					Weight 2.66
9.15	75.9	74.7	24.5	101.5	Litres 120.00
10.15	78.2	75.3	25.8	101.5	

+ .6

.0

H. P. = 25.03

H. D. = 25.03

P. M.

2.45 78.0 75.8

25.5 102.0

Weight 2.66
Litres 110.00

3.45 79.5 76.45

26.3 102.0

+ .65

.0

H. P. = 27.11

H. D. = 27.11

8.55 78.6 76.85

26.0 101.6

Weight 2.60

9.55 77.9 77.3

25.9 101.7

Litres 100.00

+ .45

+ .1

H. P. = 18.98

H. D. = 18.77

A. M.

1.45 79.7 78.

25.7 101.7

Weight 2.52
Litres 110.00

2.45 79.1 78.4

25.7 102.1

.4

+ .4

H. P. = 17.51

H. D. = 16.68

A. M.

2.55 1 gtt. of blood six days old, hypodermically.

7.05 3 gtt. " " " " " "

First Fever Day.

A. M.					Weight 2.5
9.20	75.3	73.8	24.4	102.9	Litres 97.50
10.20	76.6	74.35	24.8	103.6	

+ .55

+ .3

H. P. = 24.39

H. D. = 22.94

12.30 5 gtt. of blood 6 days old, subcutaneously.

P. M.					Weight 2.4 Litres 100.0
3.0	76.9	75.05	25.3	103.6	
4.0	77.4	75.55	25.3	104.1	
		<u>+.5</u>		<u>+.5</u>	
	H. P. = 21.85		H. D. = 20.86		

6.30 5 gtt. of blood 6 days old, subcutaneously.

9.15 Animal died, considerable diarrhoea preceding. Weight 2.34

EXP. 7.—White Rabbit.

Hunger Day.

A. M.	A. T.	C. T.	E. T.	R. T.	Weight 4.94 Litres 75.00
8.55	76.3	73.3	25.2	100.2	
9.55	75.0	74.1	24.2	100.3	
		<u>+.8</u>		<u>+.1</u>	
	H. P. = 33.78		H. D. = 33.37		
9.57	75.0	74.2	24.2	100.3	Litres 80.00
10.57	76.4	74.75	24.7	101.0	
		<u>.55</u>		<u>.7</u>	
	H. P. = 25.81		H. D. = 22.94		
11.00	76.4	74.8	24.7	101.0	Litres 60.00
12.00	78.0	75.4	25.7	100.6	
		<u>+.6</u>		<u>-.4</u>	
	H. P. = 23.40		H. D. = 25.03		
P. M.					Weight 4.92 Litres 75.00
2.00	76.7	75.45	25.2	100.6	
3.00	77.1	76.1	25.2	101.0	
		<u>+.65</u>		<u>+.4</u>	
	H. P. = 28.73		H. D. = 27.11		
4.00	77.4	76.15	25.7	100.6	Weight 4.90 Litres 75.00
5.00	78.2	76.75	26.0	101.2	
		<u>+.6</u>		<u>+.6</u>	
	H. P. = 27.47		H. D. = 25.03		

A. M.					Weight 4.88
8.05	77.0	76.8	25.4	100.9	Litres 70.00
9.05	77.9	77.4	25.9	101.0	
	H. P. = 23.94		H. D. = 27.11		

First Fever Day.

					Weight 4.78
9.00	2 gtt. of blood 17 days old, per jugular.				
9.24	74.9	73.9	24.2	101.5	Litres 72.50
10.24	75.4	74.5	24.6	102.3	
		.6		+ .8	
	H. P. = 28.20		H. D. = 25.03		

					Weight 4.78
10.30	75.5	74.5	24.6	102.3	Litres 65.00
11.30	76.1	75.2	25.0	101.8	
	H. P. = 26.81		H. D. = 29.20		

					Weight 4.76
11.40	76.2	75.3	25.0	101.8	Litres 70.00
12.40	77.0	75.95	25.3	101.2	
		+ .65		— .6	
	H. P. = 24.75		H. D. = 27.11		

P. M.					Weight 4.76
2.15	77.1	75.8	25.4	100.0	Litres 67.30
3.15	78.3	76.5	25.9	99.6	
		+ .7		— .4	
	H. P. = 27.63		H. D. = 29.20		
4.10	78.8	76.7	26.5	99.9	Litres 67.50
5.10	79.1	77.1	26.4	100.3	
				+ .3	
	H. P. = 17.74		H. D. = 16.68		
8.04.	Animal dying.			99.7	

*EXP. 8.—Cat.**Hunger Day.*

A. M.	A. T.	C. T.	E. T.	R. T.	Weight 3.32
8.45	76.8	75.25	26.2	100.6	Litres 55.0
9.45	77.2	76.0	25.6	100.0	
		+ .75		— .6	
	H. P. = 29.64		H. D. = 31.29		

FEVER.

487

9.50	77.3	76.0	25.6	100.0	Litres 52.50
10.50	78.2	76.5	26.1	99.7	
		<u>+.5</u>		<u>— .3</u>	
	H. P. =	20.04	H. D. =	20.86	
10.55	78.2	76.5	26.1	99.7	Litres 42.50
11.55	77.2	77.0	25.8	99.8	
		<u>+.5</u>		<u>+.1</u>	
	H. P. =	21.13	H. D. =	20.86	
P. M.					Weight 3.28
2.05	79.3	77.5	26.8	100.6	Litres 50.00
3.05	78.5	77.55	26.1	101.8	
		<u>+.5</u>		<u>+ 1.2</u>	
	H. P. =	24.12	H. D. =	20.86	
					Weight 3.28
4.00	79.1	77.6	26.5	101.4	Litres 57.50
5.00	78.7	78.0	26.2	101.3	
		<u>+.4</u>		<u>— .1</u>	
	H. P. =	16.42	H. D. =	16.68	
					Weight 3.26
8.05	79.2	78.5	27.3	102.4	Litres 45.00
9.05	79.4	78.7	26.5	101.8	
		<u>+.65</u>		<u>— .6</u>	
	H. P. =	25.50	H. D. =	27.11	
A. M.					Weight 3.24
7.52	76.4	74.4	25.7	100.8	Litres 115.00
8.52	74.1	74.9	23.9	100.7	
		<u>+.5</u>		<u>— .1</u>	

First Fever Day.

					Weight 3.20
9.10	2 gtt. of blood 18 days old, per jugular.				Litres 112.50
9.15	77.2	74.95	26.1	100.2	
10.15	76.2	75.5	24.7	102.0	
		<u>+.55</u>		<u>+1.8</u>	
	H. P. =	27.7	H. D. =	22.92	
					Weight 3.2
10.20	76.5	75.55	25.1	101.8	Litres 112.50
11.20	76.4	76.0	25.5	102.4	
		<u>.45</u>		<u>+ .6</u>	
	H. P. =	20.36	H. D. =	18.77	

11.25	76.4	76.0	25.5	102.6	Litres 92.40
12.25	79.1	76.45	26.1	103.5	

+ .45 + .9

H. P. = 21.14 H. D. 18.77

P. M.					Weight 3.18
2.00	82.4	76.8	28.0	104.3	Litres 77.50
3.00	83.5	77.6	28.7	102.1	

+ .8 — 2.2

H. P. 27.51 H. D. = 33.37

4.00	85.2	77.95	29.2	102.8	Weight 3.18
5.00	84.4	78.7	29.0	102.1	Litres 30.00

+ .75

8.05	84.7	79.45	28.8	103.0	Weight 3.18
9.05	83.1	80.05	27.6	101.4	Litres 72.50

+ .6 + 1.6

H. P. = 20.81 H. D. = 25.03

A. M.					Weight 3.16
7.40	80.9	80.85	27.0	101.1	Litres 163.00
8.40	82.7	81.4	28.2	100.3	

+ .55 — .8

H. P. = 20.85 H. D. = 22.94

Second Fever Day.

A. M.					Weight 3.16
8.45	2 gtt. of blood 19 days old, per jugular.				Litres 147.50
9.00	83.0	81.45	28.3	100.3	
10.00	82.0	81.80	27.8	103.5	

+ .45 .32

H. P. = 22.98 H. D. = 14.59

10.05	82.8	81.80	27.8	103.5	Weight 3.16
11.05	86.5	82.30	29.9	103.7	Litres 52.50

+ .5 + .2

H. P. = 21.38 H. D. = 20.86

FEVER.

489

11.10	86.5	82.3	29.9	103.7	Litres 142.50
P. M.					
12.10	84.4	82.78	28.9	102.3	
		<u>+.48</u>		<u>+ 1.4</u>	
	H. P. = 16.35		H. D. = 20.02		

12.40 2 gtt. of blood 19 days old, per jugular.

					Weight 3.6
1.53	84.0	82.8	29.1	105.1	Litres 70.00
2.53	84.1	83.4	29.0	104.0	
		<u>+.6</u>		<u>+ 1.1</u>	
	H. P. = 22.05		H. D. = 25.03		
4.00	84.8	83.4	29.5	102.9	Litres 75.00
5.00	85.2	84.0	29.8	101.3	
		<u>+.6</u>		<u>- 1.6</u>	
	H. P. = 20.25.		H. D. = 25.03		

5.05 2 gtt. of blood 19 days old, per jugular.

					Weight 3.00
8.00	88.0	84.5	31.5	103.8	Litres 82.50
9.00	84.9	84.9	28.5	102.0	
		<u>+.4</u>		<u>- 1.8</u>	
	H. D. = 16.68		H. P. = 12.20		
A. M.					Weight 2.98
7.30	79.0	78.45	26.9	100.6	Litres 80.00
8.30	80.0	78.75	26.8	102.1	
		<u>+.30</u>		<u>+ .5</u>	
	H. D. = 12.51		H. P. = 11.28		

EXP. 9.—Cat—tortoise-shell color.

Hunger Day.

A. M.	A. T.	C. T.	E. T.	R. T.	Weight 3.98
9.00	78.5	78.7	26.5	101.0	Litres 75.00
10.00	78.6	79.15	26.4	99.8	
		<u>+.45</u>		<u>-.2</u>	
	H. P. = 14.82		H. D. = 18.77		

10.05	78.8	79.2	26.4	99.8	Weight 3.98
11.05	80.1	79.65	26.9	99.1	Litres 65.00
		<u>+.45</u>		<u>-.7</u>	
	H. P. = 16.47		H. D. = 18.77		
11.08	80.1	79.65	26.9	99.1	Weight 3.9
12.08	80.7	80.05	27.2	99.2	Litres 135.00
		<u>+.4</u>		<u>+.1</u>	
	H. P. = 17.00		H. D. = 16.68		
P. M.					
2.10	79.6	80.0	27.1	101.4	Litres 145.00
3.10	81.4	80.5	27.5	100.1	
		<u>+.5</u>		<u>-.13</u>	
	H. P. = 16.66		H. D. = 20.86		
4.10	81.5	80.6	27.7	101.0	Weight 3.9
5.10	81.9	81.0	27.9	100.5	Litres 147.50
		<u>+.4</u>		<u>-.5</u>	
	H. P. = 15.06		H. D. = 16.68		
8.15	79.6	80.9	28.7	101.1	Weight 3.9
9.15	82.0	81.3	27.7	102.2	Litres 127.50
		<u>+.4</u>		<u>+.1</u>	
	H. P. = 17.00		H. D. = 16.68		
A. M.					
8.00	77.2	74.95	25.9	100.4	Weight 3.86
9.00	74.4	75.3	23.9	99.4	Litres 125.00
		<u>+.35</u>		<u>- 1.0</u>	
	H. P. = 13.29		H. D. = 14.60		

First Fever Day.

A. M.					Weight 3.86
9.10	2 gtt. of blood 21 days old, per jugular.				Litres 127.00
9.20	76.7	75.4	26.2	100.2	
10.20	76.7	75.9	25.2	101.3	
		<u>+.5</u>			
	H. P. = 24.70		H. D. = 20.86		

10.30	76.9	75.9	25.2	101.4	Weight 3.80
11.30	78.1	76.4	25.9	103.8	Litres 117.50
	H. P. = 28.42		H. D. = 20.86		

11.45	78.1	76.4	25.9	103.8	Weight 3.80
12.45	78.2	76.85	26.0	104.9	Litres 125.00
		+.45		+1.1	
	H. P. = 22.24		H. D. = 18.77		

P. M.					Weight 3.76
2.10	79.6	77.0	27.0	105.2	Litres 120.00
3.10	78.1	77.5	26.0	104.1	
		+.5		— 1.1	
	H. P. = 17.44		H. D. = 20.86		

4.10	81.1	77.75	27.7	103.9	Weight 3.74
5.10	78.7	78.2	26.4	103.6	Litres 125.00
	H. P. = 17.75		H. D. = 18.77		

8.15	80.1	78.4	27.0	102.6	Weight 3.74
9.15	79.0	78.8	26.1	102.7	Litres 105.00
		+.4		— .1	
	H. P. = 16.99		H. D. = 16.68		

A. M.					Weight 37.0
8.05	75.6	74.25	24.7	101.6	Litres 92.50
9.05	76.5	74.7	25.0	102.2	
		+.45		+.6	
	H. P. = 20.61		H. D. = 18.77		

Second Fever Day.

A. M.					Weight 3.7
9.12	2 gtt. of blood twenty-two days old,				per jugular.
9.15	76.3	74.7	25.	102.2	Litres 100.00
10.15	76.8	75.15	25.2	105.4	
		+.45		+ 3.2	
	H. P. = 28.59		H. D. = 18.77		

10.20	76.9	75.2	25.2	105.4	Litres 85.00
11.20	76.3	75.6	24.9	104.4	
		+.4		+ 1.0	
	H. P. = 13.61		H. D. = 16.68		

11.23	76.8	75.7	25.	104.4	Litres 75.00
12.23	77.8	76.1	25.7	103.8	

+ .5 — .6

H. P. = 19.02 H. D. = 20.80

P. M.					Weight 3.64
2.10	78.	76.15	25.9	102.3	Litres 137.00
3.10	77.6	76.6	25.4	101.7	

+ .45 — .6

H. P. = 16.97 H. D. = 18.77

4.20	78.6	76.65	26.	101.8	Litres 110.20
5.20	79.0	77.15	26.	101.7	

+ .50 — .1

H. P. = 20.56 H. D. = 20.86

8.10	77.8	77.2	25.7	101.9	Weight 3.58
9.10	78.3	77.6	25.9	102.7	Litres 120.00

+ .4 + .8

H. P. = 19.05 H. D. = 16.68

A. M.					Weight 3.56
8.00	75.	74.85	22.6	101.1	
9.00	78.	75.1	24.6	100.7	

+ .25 — .4

H. D. = 9.23 H. P. = 10.43

EXP. 10.—Cat.

Hunger Day.

A. M.					Weight 5.6
10.10	74.4	74.4	22.	101.1	Litres 77.50
11.10	78.	75.15	26.1	100.4	

.75 — .7

H. P. = 28.84 H. D. = 31.29

11.20 2 gtt. of blood seven days old, per jugular.

11.20	78.	75.2	25.6	100.4	Litres 105.00
12.20	78.	76.0	25.8	102.3	

+ .8 + .9

H. P. = 37.55 H. D. = 33.37

12.24	78.	76.	25.8	102.3	Litres 92.50
1.24	79.5	76.75	26.8	102.5	
		<u>+ .75</u>		<u>+ .2</u>	
	H. P. = 32.21		H. D. = 31.29		
1.32	79.5	76.8	26.3	102.5	Weight 5.48
2.32	79.1	77.3	26.3	104.4	Litres 70.00
		<u>+ .5</u>		<u>+ 1.9</u>	
	H. P. = 29.50		H. D. = 20.86		
2.40	79.5	77.4	26.3	104.4	Weight 5.48
3.40	79.5	78.1	26.6	104.8	Litres 105.00
		<u>+ .7</u>		<u>+ .4</u>	
	H. P. = 31.01		H. D. = 29.20		
3.45	79.5	77.1	26.6	104.8	Litres 90.00
4.45	79.8	78.8	26.8	104.0	
		<u>+ .7</u>		<u>— .8</u>	
	H. P. = 25.56		H. D. = 29.20		

Observation arrested.

The preceding experiments have been tabulated below, that they may be more readily understood and compared.

EXP. I.

	P.M. 8-9	A.M. 12.30- 1.30	8.20- 9.20	P.M. 2-3	Average Temp.	Hourly H. P.	Excess or deficit of H. P. on fever days.
H. P.	103.2	102.9	102.9	102.2	= 102.8		
	40	47	36	38			
	102.4	102.4	102.0	101.5	= 102.0		
	28	38	39	30	=	33.75	During 8-9 period, 2d fever day an excess of 2 units.
H. D.	103.9	102.5	103.8	105.8	= 104.0	=	
	121	24	24	27	=	26.00	— 7.75
	103.9	104.0	103.6	103.6	= 103.7	=	
	30	37	20	28	=	28.75	— 5.00
					Hourly H. D.		Excess or deficit of H. D. on fever days.
H. D.	45	47	39	41	=	43	
	29	39	41	31	=	35	
	27	27	22	33	=	27	— 8.0
	31	37	20	29	=	29	During 8-9 period, an increase of 2 units. — 6.0

EXP. 2.

	A.M.	P.M.		A.M.	Hourly	Excess or deficit of
	8.10-	1.45-	7.35-	12.42-	H. P.	H. P. on fever
	9.10	2.45	8.35	1.42		days.
H. P.	102.4	102.0	102.9	102.4		
	40	45	37	42		
	102.3	102.2	102.7	102.8	= 102.3	
	37	36	40	32	= 36.2	
	103.4	103.9	103.8	104.1	= 103.8	
	40	36	33	42	= 37.7	+ 1.5
	103.6	103.6	103.8	103.6	= 103.6	
	32	34	38	31	= 33.7	- 2.5
					Hourly	Excess or deficit of
					H. D.	H. D. on fever
						days.
H. D.	43	47	39	41		
	37	35	39	30	= 35.2	
	39	37	33	37	= 36.5	+ 1.3
	31	33	33	33	= 32.5	- 2.7

EXP. 3.

	P. M.				Hourly	Excess or deficit of
	12.35-	8.07-		7.55-	H. P.	H. P. on fever
	1.35	9.07	2-3	8.55		days.
H. P.	102.3	102.4	102.1	102.8	= 102.4	
	31	25	31	25	= 28.2	
	102.1	101.8	101.5	101.5	= 101.7	
	23	24	24	26	= 24.2	- 4.0
	101.0	100.9	96.8	90.6	= 97.5	
	25	17	22	18	= 20.5	- 8.2
					Hourly	Excess or deficit of
					H. D.	H. P. on fever
						days.
H. D.	33	27	31	29	= 30	
	20	27	25	29	= 25.2	- 6.8
	25	20	24	25	= 23.5	- 6.5

EXP. 4.

	P. M.				Hourly	Excess or deficit of
	1.44-	9.20-		8.55-	H. P.	H. P. on fever
	2.44	10.20	3-4	9.55		days.
H. P.	100.9	101.3	101.0	101.8	= 101.2	
	27	23.2	19	25	= 23	
	102.1	101.4	101.4	100.8	= 101.4	
	23.2	17.6	22	20	= 20.7	
	102.3	100.2	101.3	102.1	= 101.4	
	22.9	22	23	20	= 21.9	+ 1.2
	101.7	99.1	98.4	97.1	= 99.0	
	19	17	20	25	= 20.2	- .5

					Hourly H. D.	Excess or deficit of H. D. on fever days.
H. D.	25	25	18	22	= 22	+ 2
	22	18	25	20	= 21	
	22	20	25	25	= 23	
	22	14	22	25	= 20.7	

EXP. 5.

					Hourly H. P.	Excess or deficit of H. P. on fever days.
A. M.						
8.10-						
9-10						
H. P.	101.9	102.5	102.1	102.1		+ 1
	31	35	28	27		
	101.7	101.4	101.9	102.0	= 101.7	
	18	22			= 20	
	102	103.6			= 102.8	+ 1
	17	25			= 21	
					Hourly H. D.	Excess or deficit on fever days.
H. D.	27	31	27	27	=	- 5
	25	27			= 26	
	22	20			= 21	

EXP. 6.

					Hourly H. P.	Excess or deficit of H. P.
A. M.						
8.55-						
9.55						
Hunger Day.	100.2	100.3	101.0	100.6	100.9	= 100.6
	33	25	23	28	27	= 27
	101.5	102.3	101.8	100	99.9	= 100.9
	28	26	24	27	17	= 23
						- 4
					Hourly H. D.	Excess or deficit of H. D.
H. D.	33	22	25	27	25	= 26
	25	29	27	29	16	= 25

EXP. 7.

					Hourly H. P.	Excess or deficit of H. P.
A. M.						
8.45-						
9.45						
Hunger Day.	100.6	100	99.7	100.6	101.4	102.4
	29	20	21	24	16	25
	100.2	102	102.6	104.3	102.8	103
	27	20	21	27	29	20
H. P.	100.3	103.5	103.7	105.1	102.9	103.8
	22	21	16	22	20	12
						11
						= 18
						+ 1.2
						- 4.2
					Hourly H. D.	Excess or deficit of H. D.
H. D.	31	20	20	20	16	27
	22	18	18	33	31	25
	14	20	20	25	25	16
						20
						= 22
						22
						= 24.1
						= 18
						+ 2.1
						- .4

EXP. 8.

		A. M.	10.05-	11.08-	2.10-	4.10-	8.15-	8-9	Hourly H. P.	Excess or deficit of H. P.
		9-10	11.05	12.08	3.10	5.10	9.15			
H. P.	{	101.	99.8	99.1	101.4	101	101.1	100.4 =	100.5	
		14	16	17	16	15	17	13 =	15.4	
		100.2	101.4	103.8	105.2	103.9	102.6	101.6 =	102.6	
		24	28	22	17	17	16	20 =	20.5	+ 5.1
		102.2	105.4	101.4	102.3	101.8	101.9	101.1 =	102.7	
		28	13	19	16	20	19	9 =	17.8	+ 2.4
									Hourly H. P.	Excess or deficit of H. D.
H. D.	{	18	18	16	20	16	16	14 =	16.8	
		20	20	18	20	18	16	18 =	17.1	+ 3
		18	16	20	18	20	16	10 =	16.5	- 3

EXP. 10.—Cat.

First Hunger Day.

A. M.	A. T.	C. T.	E. T.	R. T.	Weight 5.6 Litres 77.5
10.10	70.	74.4	22.1	101.1	
11.10	78.	75.15	26.1	100.4	
H. P. = 28.84		H. D. = 31.29			
11.19	2 gtt. of putrid blood seven days old, per jugular.				
11.20	78.	75.2	25.6	100.4	Litres 105.0
12.20	78.	76.0	25.8	102.3	
H. P. = 37.55		H. D. = 33.37			
12.24	78.	76.	25.8	102.3	Litres 92.50
12.24	79.5	76.75	26.8	102.5	
H. P. = 32.21		H. D. = 31.29			
1.32	79.5	76.8	26.3	102.5	Weight 5.48
2.32	79.1	77.3	26.2	104.4	Litres 70.0
H. P. = 29.50		H. D. = 20.86			
2.40	79.5	77.4	26.3	104.4	Litres 106.0
3.40	79.5	78.1	26.6	104.8	
H. P. = 31.01		H. D. = 29.20			
3.45	79.5	78.1	26.6	104.8	Litres 90.0
4.45	79.8	78.8	26.8	104.0	
H. P. = 25.56		H. D. = 29.20			

EXP. 11.—Rabbit. Cortex cerebri removed.

P. M.	R. T.
3.34	101.8
3.35 2 gtt. of putrid blood, per jugular.	
4.05	101.6
5.20	105.4
5.55	102.4
7.00	101.6

Second Day.

A. M.	
8.45	103.0
8.47 1 gtt. of blood, per jugular.	
9.10	102.8
9.33	103.0
10.07	103.4
11.35	104.1
12.07	103.0

Upon autopsy, a large part of the cortex was found to be destroyed.

EXP. 12.—Rabbit. Cortex removed.

P. M.	R. T.
1.24	103.7
1.45 2 gtt. of putrid blood, per jugular.	
1.54	106.1
2.42	105.7
4.00	106.0

At autopsy, a large part of cortex cerebri destroyed.

EXP. 13.—Rabbit. Anterior end of corpora striata destroyed.

P. M.	R. T.
1.40	104.8
2.15 $\frac{1}{4}$ gtt. of putrid blood, per jugular.	
2.45 $\frac{1}{2}$ gtt. " " " " " "	105.1
2.47	106.3
4.15	105.3
5.00	105.2
6.20	104.8

EXP. 14.—Rabbit. Corpora striata removed.

P. M.	R. T.
4.45	103.4
5.35 $\frac{1}{4}$ gtt. of putrid blood, per jugular.	103.6
6.00	103.8
6.20	103.6
6.50 $\frac{1}{2}$ gtt. of putrid blood, per jugular	103.9
7.05	103.6
7.25	102.9
7.45	102.0
8.10	102.4

EXP. 15.—Rabbit. Anterior ends of corpora striata removed.

P. M.	R. T.
12.45.....	103.2
1.42 ¼ gtt. of putrid blood, per jugular.	
2.10.....	103.4
3.00.....	105.2
3.25.....	106.4
4.10 ..	106.6
.....	106.2

EXP. 16.—Rabbit. Corpora striata removed.

A. M.	R. T.
11.50.....	103.8
12.40 ¼ gtt. of putrid blood, per jugular.....	102.8
1.05.....	102.1

EXP. 17.—Rabbit. Corpora striata removed.

P. M.	R. T.
5.42 ..	103.9
5.49 1 gtt. of putrid blood, per jugular.	
6.15.....	102.8
6.45 ..	102.6
8.10 ..	100.8

CASE OF SYPHILOMA OF THE CORD OF THE CAUDA EQUINA—DEATH FROM DIFFUSE CENTRAL MYELITIS.

BY WILLIAM OSLER, M. D.

Professor of Medicine, Johns Hopkins University, Baltimore.

THE following case which was under the care of Dr. S. Weir Mitchell, at the Infirmary for Nervous Diseases, Philadelphia, presents many points of clinical and anatomical interest.

Clinical Summary.—Chronic alcoholism, history of syphilis. For nine months pains in the legs, particularly in the left, which wasted rapidly, and presented vaso-motor changes. Pains in the arms, especially the right; no wasting, and, on admission arms of equal strength. About two months before death loss of control of bladder and rectum. Within the last month of life loss of power in the right arm, with pains; partial loss of power in the left arm with marked inco-ordination, complete paralysis of the left leg, gradual loss of power in the right. Development of bed sores. Arthritis in knees and ankles. Towards the close of life, high fever with delirium.

Anatomical Summary.—Gumma in antero-lateral columns of cervical cord opposite the right fourth anterior nerve root. Gummata involving the third, fourth and fifth anterior sacral nerve roots, and the second and third posterior sacral roots on the left side. Ascending degeneration of the left posterior median column. Central myelitis. Partial atrophy of the sciatic nerves.

A. B., æt. 42, lawyer, admitted February 5, 1888. Family history good. Had been a hard drinker for years and had smoked and chewed to excess. He had gonorrhœa four times, and a soft chancre but no history of secondaries could be obtained.

In 1876 he had delirium tremens.

About April, 1887, he began to have sharp, shooting pains in the arms and legs. They came on suddenly, were stab-like in character, lasting only a moment and then passing off. No definite regions in the arms and legs were involved. He also had dull pains in the back of the head and neck. These troubled him more or less throughout the summer, but he could get about fairly well. Towards the second week in October the pains began to be more severe in the left leg; they were thought to be rheumatic in character. About the twenty-fourth of October, his suffering was so great that he was confined to bed. By November 5th he could scarcely walk. The pain began in the right arm and shoulder, the right leg also was painful and weak. There was no redness or swelling of the knees, but the left foot and ankle would get red and swollen, almost purple. The left leg wasted rapidly and for a time he lost sensation in the legs completely. The left arm remained unaffected. About a month before his admission he lost control of his bowels and had a constant desire to urinate. He had to use the catheter for several weeks.

The following notes of his condition were taken on admission by Dr. Burr, Resident Physician.

"He can stand a little with the aid of a chair and he can flex and extend the right knee and hip. He cannot move the left leg, the knee of which is swollen. He has very little pain, none in the right leg. The wasting of the left leg is marked. The knee-jerk is present on the right side but on the left side it is obtained with difficulty. On the right side cremasteric reflex is present, absent on the left. Abdominal reflex present on both sides. No tender spots over spine; bed sores on the coccyx and on the left buttock; has pain in the shin bones and in the groin at night. The arms show almost equal strength. The dynamometer registers 115 for the right hand and 120 for the left.

For two weeks he seemed to be in much the same state though in rather less pain. Towards the end of the month the ankles and knees became more swollen; the bed sores had healed.

On March 16th. the note is as follows: "Has been unable to move the right knee or thigh since yesterday; the swelling has subsided in the knee but the ankle remains swollen; the fingers of the left hand have been numb since yesterday; pain along the inner side of both arms and at the points of the elbow; pain in the left shoulder for several days; right hand is powerless; fingers held flexed in palm; can move the right shoulder: is losing power in the left arm



and hand; movements are distinctly ataxic; there is pain on spine over the seventh cervical vertebræ, worse on pressure."

From the 17th to the 20th the temperature rose gradually, reaching 102° , and at this date he lost sensation in the ulnar distribution of both hands.

21st.—Delirious, but can be easily roused, when he will talk rationally for a few minutes; tongue red, dry and coated; pupils contracted; pulse rapid and feeble; gangrenous bullæ on the outer side of heel; temperature rose this morning to 105° and remained high all the morning. At 2 P. M. it reached 106.8° . Cold sponging and antipyrin reduced it to 102° by evening.

22d.—The delirium persists and bed sores have again appeared on the sacrum; the scrotum is œdematous; he has difficulty in swallowing; the breathing is diaphragmatic; does not complain of pain; temperature, to-day remained below 104° .

23d.—General condition unchanged; is unconscious and is roused with difficulty; morning temperature was 102° rising gradually during the afternoon till it reached 105.6° at 7 P. M.; at 10 P. M., it was 106.8°

24th —Low, delirious fever continues, reaching at 12 M. 107° and continued elevated during the afternoon. At 10 P. M., the rectal temperature was 108° ; at 12:30 A. M., 108.4° ; at 2 A. M., 108.8° ; at 3 A. M., 109.4° . See chart.

Death occurred at 4 A. M.

Post-mortem, five hours after death.

Body emaciated, left leg smaller than the right; scrotum œdematous; superficial gangrenous bullæ on each heel; recent bed sores on sacrum.

The skull cap was removed with difficulty, as there were strong adhesions to dura.

Longitudinal sinus contains blood. Parts at the base of skull normal; cortical arachnoid, opaque. Pachionian granulations abundant and large; pia mater turbid, strips off readily from hemisphere, but is somewhat œdematous. Convolutions look healthy, and the gray matter is of a rosy pink color; white substance moist, with very few bleeding points; lateral ventricles look dry; third and fourth ventricles present no changes; in the latter, the vessels just above the acoustic striæ are a little congested.

Section of the ganglia at the base show no foci of disease; pons and medulla symmetrical; no descending lesions.

Cerebellum normal.

Spinal Cord.—Dura mater natural looking, nowhere adherent except at the anterior part of cervical enlargement; no sub-dural exudation; arachnoid thin and clear. On the right half of the cervical enlargement the dura is attached to the arachnoid and to the pia over an area the size of a split pea. There is here a firm solid mass in the cord, not producing any special deformity, but appearing extern-

ally as a grayish region, situated between the anterior roots of the third, fourth, and fifth cervical nerves. The fourth is involved in the adhesion of the dura. The anterior roots are not involved, nor does the adhesion of the dura extend laterally beneath the dentated ligament. The grayish translucent appearance of the mass extends for about a line beyond the posterior median fissure. Vertically it is about one-third of an inch in length.

Fresh sections were made at the following points:

Second Cervical.—Interior soft, but outlines of gray matter distinct. The left column of Goll has a grayish-white translucency.

Sixth Cervical.—Gray matter has lost its firm appearance, and is very soft and reddish in color.

Seventh Cervical.—Central softening still apparent. Cornua not distinguishable.

Second Dorsal.—Gray matter more natural looking.

Eleventh Dorsal.—Outline of gray matter quite distinct. There is a marked degeneration of the left postero-median fasciculus.

The cauda equina presents the following alterations: The three last anterior nerve roots leaving the conus medullaris are involved in a gummous growth the size of a bean, into which pass also the posterior roots of the second and third sacral nerves of the left side. They are involved about two inches from the cord. Lower in the canal there are two or three small fibres, which present slight tuberos enlargements.

The tumor of the cord varies in transverse diameter from three-eighths to one-quarter of an inch in diameter; it is completely within the cord, the symmetry of which is not materially altered (Fig. 1). In shape, above and below, it is rounded; in the middle, more ovoid. The vertical extent is not quite half an inch. At a limited region the dura is adherent to the pia, which membrane, at this point, is distinctly thickened. With a low power it is seen that the growth occupies the right antero-lateral region, destroying and pushing aside the anterior cornu, displacing the antero-median fissure and pushing back the posterior

cornu. In the upper part of the growth, the outlines of the gray matter of the left side and of the right posterior horn are well seen. In the middle portion they are much less distinct; and here the growth reaches so far over that it is only one-eighth of an inch from the left lateral margin of the cord. The growth is firm, not encapsulated, and sections in carmine stain of a deep red color. The greater portion of the mass is made up of a dense fibro-caseous tissue, devoid of cell-elements, and through which passes a



Fig. 1.—Gumma of cervical cord opposite fourth nerve root.

number of blood-vessels, some of which are obliterated, some free. At the periphery, there is marked cell proliferation, particularly towards the gray matter. This is also very distinct in the anterior median fissure. The anterior spinal artery is involved at the edge of the growth, and the adventitia encircled in three-fourths of its extent. The intima is greatly thickened, and the cell elements look much swollen. In the adherent dura, which is not thickened, there are amyloid bodies. The gray matter looks swollen; at the upper portion of the tumor area, the large cells are distinct, but the nuclei do not stain well in carmine. In the middle and lower portions of the affected

regions, the nerve cells are much less distinct, and there is extensive infiltration with leucocytes, particularly in the neighborhood of the vessels.

In the white matter the axis cylinders everywhere stain in the carmine, but the neuroglia looks swollen, and has very indistinct outlines.

The cervical cord, above the gumma, stains well in both carmine and by Wiegert method. The gray matter is distinct, and the nerve cells look somewhat swollen; their nuclei stain well.



Fig. 2.—Lumbar cord, showing degeneration of the left posterior column.

The tumor of the cauda has matted the nerve roots together, and sections in hæmatoxylin and eosin show large areas of indifferent tissue stained red, surrounded by zones of actively poliferating connective tissue, the cells of which stain deeply in the hæmatoxylin. In the central caseo-fibrous regions the outlines of the nerve bundles can be seen, and, in places, numerous irregular areas, lighter in color, closely set together, which represent the degenerating nerve fibre with their medullary sheaths pale, and many of the axis cylinders stained.

The degeneration of the left posterior column is interesting. In the lumbar cord it involves a wide area, chiefly in the root zone, not reaching the median surface or the posterior, except close to the nerve root (Fig. 2). In the dorsal cord (Fig. 3) the root zone is not involved, and the whole column of Goll is affected except a narrow wedge.

In the region of the tumor the degeneration does not reach so close to the posterior margin (Fig. 1).



Fig. 3.—Dorsal cord. Descending degeneration of left columns of Goll.

The left sciatic is extensively degenerated. In the right there are two or three bundles in which atrophy is apparent. By Weigert's method the contrast is very striking, as shown in Figs. 4 and 5.



Fig. 4.—Left sciatic nerve. Cross section.

The early pains, at first in the arms and legs, then chiefly in the right arm; the wasting, weakness, and gradually total paralysis of the left leg; the slow onset of the paralysis of the right arm with paresis of the left, find their explanation in the progressive growth of the tumor in the cervical cord. The involvement of the anterior sacral roots was responsible in part for the loss of power in the legs,

but the early affection of the left with rapid wasting was undoubtedly the result of the cord lesion.

The accurate localization of the lesions in the cauda equina makes a consideration of the symptoms produced by them of some importance. Unfortunately, there is no note upon sensation in the perineal and gluteal regions, but for two months previous to death there was loss of control of the bladder and rectum. We can, I think, look upon this case as confirming the view that the ano-vesical centres are in the

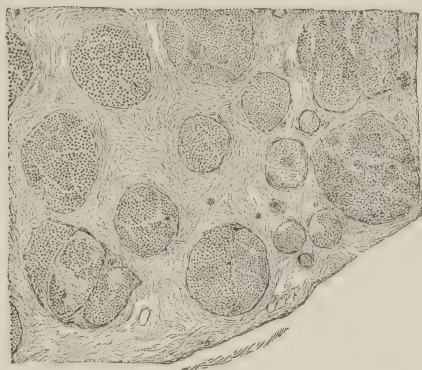


Fig. 5.—Portion of cross section of right sciatic nerve.

sacral, not in the lumbar segments of the cord. The disturbance in the reflex arc was here chiefly in the efferent branches involved in the third, fourth and fifth cords. It will be remembered that of the afferent branches only the second and third sacral roots were involved.

A third point of interest is the ascending degeneration in the left column of Goll due to the lesion in the second and third posterior sacral roots, and, in part also, undoubtedly, to extensive disease of the left sciatic nerve. As is shown in the figures, the distribution of the sclerosis presented the well-known variations in passing from the lumbar to the cervical cord.

Lastly, the case offers an excellent illustration of the chief symptoms of acute central myelitis, particularly in the high temperature, the arthritic disturbances and the marked trophic changes, as shown in the rapid development of bed sores.

CLINICAL HISTORY AND AUTOPSY OF A CASE
OF GENERAL PARESIS OF NINE YEARS'
DURATION.

From the Clinical Records of the Bloomingdale Asylum, New York.

By WILLIAM NOYES, M. D.,

Second Assistant Physician.

PATIENT, Mr. J., admitted to Bloomingdale Asylum, April 21, 1878; age, thirty-six; born in Germany; married; natural disposition amiable; sanguine and enthusiastic; good intellect; common school education; his habits were temperate and regular; smoked, but did not drink; had had syphilis; no hereditary taint known; first attack; patient had been a successful business man, but failed about a year before admission, and then set to work earnestly to retrieve his fortunes; about the first of July, 1877, he began to be dictatorial and irritable, entertained extravagant delusions, and made foolish and lavish purchases; these symptoms became more marked, and he grew boisterous and indecent in language and manifested great sexual excitement.

About September 1st his articulation was noticed to be impaired. On September 7th he was sent to a small private institution near New York, where he remained until March 1, 1878, and was then taken home on trial, but gave much trouble. Has gained flesh markedly of late. On admission to Bloomingdale he showed tremulousness of extremities, unequal and dilated pupils, marked difficulty in articulation, and characteristic feeling of satisfaction and health.

May 4th.—Became confused mentally, so that he did not appreciate where he was; thought his wife somewhere near him; was noisy, shouting and pounding on his door; thinks he lacks various organs, and asks the attendants to

bring a new heart, lungs, and brain ; power of articulation seems less impaired ; pupils widely dilated, unequal, irregular, and very changeable in size.

May 9th.—Condition but little changed ; eats well, and is in good physical condition ; frequently disarranges and tears his clothing ; is excited by presence of others ; much of the time repeats in Hebrew what seems to be a religious chant, accompanying it with stately gestures, and seems to see beautiful and grand visions ; masturbates considerably.

July 16th.—His dementia has increased very decidedly recently ; rubs his head a great deal or strikes it, sometimes violently, as though it caused him great discomfort ; no physical symptoms of localized cerebral lesion can be detected.

August 20th.—About ten days since, during a period of unusual excitement, in which he beat his head a great deal, a hæmatoma formed in the upper portion of each ear ; often very noisy and destructive of clothing.

November 12th.—There has been a slight abatement of excitement during the past three weeks ; occasionally will be quiet and seem interested in reading, but at times is as excited and boisterous as ever.

January 21, 1879.—Slight gradual mental and physical failure, but not marked ; paretic symptoms have not increased.

May 12th.—Little change ; no advance of paretic symptoms ; is more demented, but slightly less violent in excitement ; general physical condition good ; often untidy, and somewhat destructive ; often beats his head violently and rubs it.

August 18th.—No change except gradual failure, mental and physical ; excitement less active ; has insatiable thirst, and will drink great quantities of water, even his own urine, if allowed.

December 28th.—Is very greatly demented ; very untidy ; if questioned or spoken to, answers with a grunt.

June 19, 1880.—More demented and untidy, otherwise unchanged.

August 30th.—Very stout and quite muscular ; nervous symptoms do not appear to progress.

December 21st.—Remains without apparent change, mentally or physically.

February 7, 1881.—Continues very untidy ; when spoken to, answers with a succession of grunts.

February 6, 1882.—Walks about the hall carrying a small music-box, for which he displays a remarkable affection ; unable to speak ; eats well, but is occasionally restless at night.

May 1st.—Body well nourished ; does not speak ; keeps his mouth very tightly closed, and has worn his teeth very much by incessant grinding.

September 27th.—Continues to gain in weight.

October 7th.—Does not speak, and appears unable to do so, but expresses his likes and dislikes by signs ; is much attached to his music-box, and shows marked delight when it is wound up and begins playing.

November 30th.—Eats with apparent enjoyment ; continues to take great interest in the music-box, and is entirely contented when it is playing ; uses it for a head rest.

1883.—Condition unchanged.

November, 1884.—Never speaks, but still enjoys his music-box.

March 9, 1885.—Has begun again his old habit of slapping his head and face ; eats and drinks voraciously, if permitted.

June 24th.—Is growing weaker ; this morning fell down a flight of stairs, falling about three feet, but with no serious injury.

August 28th.—Ataxia has increased so much that he cannot rise from his bed or from the floor if he falls, but is still able to walk if placed on his feet.

September 3d.—Grows more unsteady on his feet, and now lies down most of the time.

September 15th.—Became much excited ; got up during the night and came out of his room several times.

September 16th.—Is very helpless, and cannot rise from his bed or from the couch that he occupies in the yard.

October 26th.—Swallowed a horse-chestnut given him by another patient ; the body was extracted in time to prevent the patient from choking, but he had become much cyanosed.

March, 1886.—But little change during the winter except a gradually progressive weakness ; not yet confined to bed, but sits up in an easy-chair.

April 8th.—Had a convulsive attack this morning ; his eyes and mouth twitched considerably ; the attack passed off without any serious disturbances.

April 9th.—No return of the convulsion, and is apparently as well as usual.

April 10th.—Face twitched somewhat at midnight, but as well as usual in the morning.

April 13th.—Slight twitching of left side.

April 15th.—Somewhat tremulous, but no further signs of a convulsion.

April 16th.—Temperature of 101.2° at night, and 100° in the morning ; no cause apparent.

April 21st.—Sitting up for the first time in several days.

April 23d.—Slight twitching of right side of face.

April 24th.—Twitching somewhat increased.

April 25th.—Twitching has ceased.

April 27th.—Slight twitching of face again.

May 5th.—Slight twitching of face again.

May 8th.—Right leg twitching somewhat.

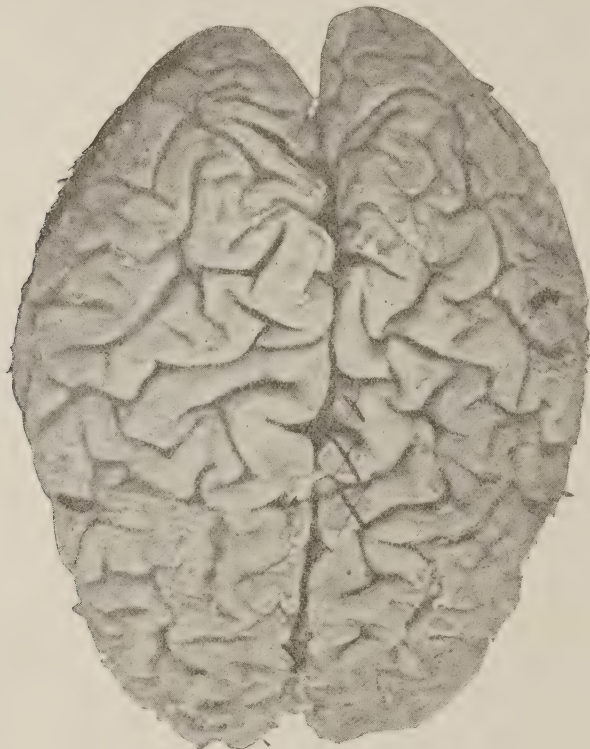
May 13th.—Had a convulsive attack at 9 P. M., with general twitching until morning ; temperature, 103° at 2 A. M. ; twitching had ceased by morning of May 14th.

May 15th.—Twitching continued through last night, and a few convulsions toward morning ; is weaker than at any time before. (Mustard sinapisms were applied to his abdomen during the convulsive attacks, and injections of morphia were given when demanded by the severity of the twitchings.)

May 16th.—Continued about the same through the day ; dry cups to his spine and neck in the afternoon and mustard to his abdomen ; appeared about the same at 5.15 P. M., but died suddenly at 5.30 P. M.

Autopsy, May 17, 1886, in presence of Dr. Sanger Brown, first assistant physician, and Dr. B. Sachs.

Body greatly emaciated. No anomalies of head or trunk. Thoracic organs normal, except a small amount of hypostatic pneumonia in lower lobe of right lung. Head well formed. Skull of ordinary thickness. Dura mater thickened and opaque. Pia mater thickened and firm, and adherent to surface of brain, especially over the frontal lobes.

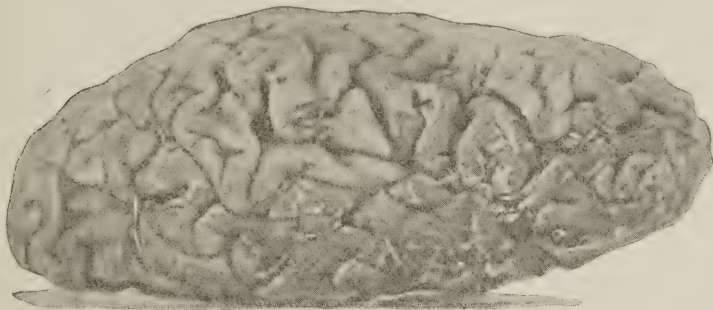


View of upper surface of the brain.

The convolutions, after the removal of the pia, presented in a high degree the typical "worm-eaten" appearance of paresis. The convolutions were markedly atrophied and shrunken, and the sulci wide and gaping.

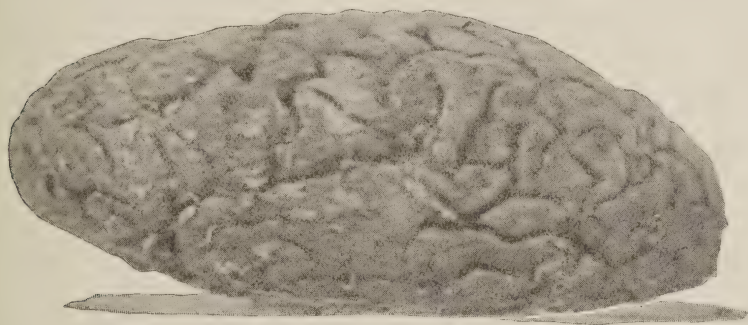
The accompanying photographs were taken after the brain had been hardened thoroughly in Müller's fluid, and show all these characteristics. In a few places on the top

of the brain the cortex was torn a little mechanically in the manipulation of photographing, on account of the extreme brittleness after the prolonged hardening ; these spots on the superior and occipital portions of the brain are easily



Lateral view of right hemisphere.

distinguishable from the "worm-eaten" portions of the frontal lobes, where the tearing of the cortex was due to the adherent pia. Microscopic examination will be reported later.



Lateral view of left hemisphere.

Regarding the clinical history of the case, it will be noted that the disease pursued an unusually quiet course ; dementia came on early, and there were no attacks of violence. There were also no congestive attacks until shortly before death, and these were of a mild character.

Mendel, in his "*Die progressive Paralyse der Irren*" edition of 1880, p, 270), found from his observations four cases of from eight to ten years' duration, and one case of sixteen years' duration, the latter of the ascending type.

PERISCOPE.

PERSISTENT SPASM OF THE LEVATOR ANGULI SCAPULÆ MUSCLE.

From Transactions of American Neurological Association.

MONOSPASM, or spasm of a limited number of muscular fibres or groups of muscles, like monalgia or pain restricted to single trunk, branch or centre of a sensory nerve is equally significant and valuable to the neurologist, for diagnostic purposes, with diffuse pain or generally distributed convulsive movement. It is significant and interesting by contrast and for comparison and physiological study as well as for clinical diagnostic purposes.

In our study for neurological phenomena, we are quite familiar with *mono* as well as with the multiple or general spasm, with monomania as well as with general insanity, with localized, as well as with general neuralgia. In fact, manifestation of these in localities and in part is more common than in general.

But certain forms of chronic monospasm are as infrequent as are that limited and restricted psychical painful disease which we might designate as monomelancholia, a phase of a psychiatric disorder which has sometimes fallen under my own personal observation and been described in other terms by good psychiatric authority, but which may be called a comparatively rare form of mental disease.

The spasmodic tabetic crises (laryngeal æsophageal, cardiac, gastric, etc.), possess a painful and peculiar interest to the neurologist, which we place in juxtaposition for interesting differential study, with the more evanescent and not so discouragingly significant phenomena of hysteria and the grave states of atrophic and spastic paralysis.

No part of the muscular system innervated by the cerebro-spino-neural mechanism is free from the possibility of spasms ; from the muscles of the eye to those of the scrotum or testicle, though we are clinically and by physiological experiment much more familiar with spasm in some parts of the organism than in others.

The spasmodic phenomena of tetanus, of tabes, of hystero-epilepsy, of tetany (or tetanilla, as Ross prefers to call it, and which is more exactly expressive), of catalepsy, of Thommssen's disease, of the eclampsias (toxic, anti-toxic, thermic or febrile, idiopathic febrile, asphyiate, reflex, etc.), and the monospasms and unilateral convulsions of cerebral cortex, irritation, etc., remind us how omnipresent and universal, for cause, spasms may be in the organism.

Trismus, torti-collis, main en griffe, angina pectoris, Bell's palsy, occulo-motor monospasm and the graphospasms, are the professional hyperkinésies that have become classical through oft recognition and exact description in the literature, but there is a region of the neck and shoulder which I have seen under the influence of prolonged spasm in an extremely young subject—a child of two years—which I have never seen before or heard described by any *confrère* in the study of neuriatry or psychiatry, nor is generally described in the books on neurology. Ross, however, has mentioned it as a spasm of the levator anguli scapulæ, "the upper and inner angle of the scapula being strongly elevated, the head slightly inclined to the same side, the shoulder drawn forward, the supra-clavicular form increased in depth, the contracted muscle projecting distinctly beneath the anterior border of the trapezius," giving the appearance in a young child, of congenital deficiency, as well as deformity by displacement of the scapula. The displaced scapula appears at first sight much smaller than its compliment on the opposite side, though by actual measurement there appears no difference.

The child is two years old. The deformity appeared about six months ago, after a spell of fever, probably convulso-spinal fever, from the mother's description. The father's history could not be accurately learned, but he is

an apparantly healthy laboring man ; drinks, but not an inebriate. The mother brought the child to Dr. Harry Hodgen, from whose hands the case was passed to me. Dr. Hodgen's acknowledged skill in orthopedic diagnosis not enabling him to detect any joint lesion, or any tendious trouble remediable by operation.

The cause of this spasm is in my judgment, cervical spino-meningeal irritation at the origins of the brachial plexus, from which innervation of the levator anguli scapulae and contiguous muscles of the shoulder-blade is derived.

This meningeal irritation is probably the legitimate sequence of a previous spinal meningitis.

C. HUGHES, M.D., St. Louis.

Reports of Asylums.

THE DIFFICULTY OF PROCURING AND RETAINING EFFICIENT ATTENDANTS IN HOSPITALS FOR THE INSANE.

Dr. H. Rooke Ley, Medical Superintendent of the Prestwich County Lunatic Asylum, England, in his report for 1888, refers to the difficulty of securing and retaining the services of a staff of efficient and trustworthy attendants as one of the chief embarrassments encountered in the organization and government of institutions for the insane. Dr. Ley correctly voices the observation and experience of a majority of superintendents of asylums, the world over, when he says :

“ The difficulty is not alone confined to England, where labor is scarce, but throughout the world, wherever Asylums exist, the great problem in their administration has been the maintenance of an efficient and reliable Nursing Staff. To a certain extent our General Hospitals labored under the same difficulty some twenty-five years ago, but since then sick nursing has been taken up as a profession, and is now established on a tolerably sure and safe basis. Those cultured and refined women who, whether as members of an organized society or as individuals, have devoted themselves to the care of the sick as a labor of love, with no reward but that of serving God and their fellow creatures, have looked askance upon Asylum service, although to such this vocation offers an illimitable field for the exercise of their benevolent sympathies. If some of these devoted women or their followers could be induced to set an example, and take service in our Lunatic Asylums, their presence would no doubt exercise an influence both important and salutary. But service in Asylums has never become, and is not likely to become, popular in the same way as Hospital service has become popular of late years, for nursing the insane is a very practical and very serious business, involving hard work, numerous risks, great responsibility, affording little scope for sentiment or dilettanteism. It is a calling, also, that leads to nothing outside of an Asylum, the demand for qualified attendants being small, as compar-

atively few insane persons are treated at their own homes. As it is, the Asylum has to do the best it can, and although applicants for the position of nurses and attendants are plentiful enough—for every failure in other careers considers himself or herself competent to attend upon the insane—only a small percentage of those who apply possess the moral qualifications and necessary intelligence to warrant their engagement. When such are found, and well trained, the competition with kindred Institutions is so keen that there is always the fear of the attendant transferring his services elsewhere. No doubt much of the unrest of attendants is due to causes, some of which are to a certain extent removable. Apart from the nature of the duties which they have to perform, and which must always be arduous, trying, and often disagreeable, the hours of work are long, living night and day in Wards with the insane, their rest is apt to be disturbed, and the calls made upon their physical and mental energies are greater than many can bear.”

With a view of remedying these drawbacks, in the institution so ably presided over by Dr. Ley, the staff of night nurses has been largely increased, so as to admit of a better division of labor and thus entirely dispense with the services of the day attendants after a certain hour in the evening, when they are privileged to retire to quarters quite apart from the Wards, after the work of the day is done, “thus enabling them to obtain that relaxation by day and that rest at night so essential to the proper performance of their duties.” The apartments for attendants are roomy, well furnished and arranged to insure the comfort of their occupants. This is a step in the right direction, and one that must inevitably tend to prove the quality and stability of the service by the attraction of a higher class of applicants for admission thereto.

Anent this subject, the annual report of the State Asylum for Insane Criminals (1888), at Auburn, N. Y., contains the following :

“The position of an asylum attendant is by no means an enticing one ; and inasmuch as the degree of success to be attained in the treatment of the insane, in asylums, depends to a considerable extent on the efficiency and fidelity of

attendants, who necessarily have the immediate care of the patients, it would seem to be both wise and economical to make such reasonable provisions for their comfort, in the matter of privileges, compensation, food and quarters, as will induce those of approved adaptability to remain in the service after they shall have acquired that amount of experience and training which alone fits them for the delicate and important duties they are required to perform. It has been our aim in recent years to improve the status of attendants in these several respects, so far as structural arrangements of the institution and the necessities of the service would permit ; and, from the apparent improvement in the quality of the services rendered by them, we are led to believe that the effort has yielded good results."

Again, the superintendent of that institution, in the twenty-fourth annual report, after referring at length to the duties and trials of attendants, says :

"From the foregoing it will be seen that 'the good attendant' must necessarily possess superior and peculiar qualities of both head and heart to enable him to acceptably discharge the numerous and trying duties incident to his position. He must be prepared to perform offices of a repulsive nature and which are not seldom rendered difficult and more repulsive by reason of the resistance and opposition which insane persons frequently offer to efforts in their behalf. It not infrequently happens, on unlocking them in the morning, that several patients on a ward are found to have soiled not only themselves, but their bedding, and even the floors and walls of their rooms, with their excrement. Such patients must be bathed, their under-clothing and bedding changed, and their rooms cleansed and put in order before breakfast, a kind of work which, to say the least, is not conducive to appetite ; again, attendants must not only consent to serve long hours for comparatively small wages, but must exercise control of temper under the widest range of provocation, embracing insulting and abusive language, groundless accusations and even assaults, on the part of patients, endangering both life and limb. They are constantly reminded by the medical officers that to strike a patient is one of the gravest offences, and which,

if proven against them, will lead to instant dismissal with forfeiture of a month's pay; that they must constantly bear in mind that patients are irresponsible beings, against whom they must harbor no ill will, but must feel and act as kindly toward the most disagreeable patient as they do toward those that are pleasant and agreeable. While it occasionally happens that an unsuitable person succeeds in obtaining employment as an attendant, despite the rigid scrutiny to which applicants are subjected, and a constant endeavor to select from among those who seek to enter the service only such as are apparently possessed of the qualifications which are necessary to enable them to discharge the arduous, trying, and often thankless duties that fall to the lot of an asylum attendant, it may truthfully be said that instances of brutality, on the part of attendants, are exceptional, infrequent, and of short duration. On the other hand, with equal truthfulness, be said that the majority of asylum attendants are painstaking, faithful to their trusts, and merit public confidence and esteem, instead of the distrust, misrepresentation and hasty condemnation which are so readily, and often wrongfully, bestowed upon them. Instances of heroic, self-sacrificing devotion to duty on the part of attendants are frequently witnessed by the medical officers of asylums, while, from the nature of things, the general public can have no adequate appreciation of the perplexities and trials that must daily be encountered by attendants in the discharge of their duties."—*Carlos F. MacDonald.*

**Which is the Most Powerful ?
and the Most Reliable ?
of All Pepsins ■**

Merck's Pepsin 1:2000 !

— SCALE OR POWDER —

➡ SEE "MERCK'S INDEX," PAGES 106 AND 167 ➡

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

REPORT OF CASES OF CHRONIC DELUSIONAL
INSANITY—PARANOIA.

By MATTHEW D. FIELD, M.D.,¹

NEW YORK.

CASE I.—W. E. L., male, aged 29, a photographer, was committed for examination as to his sanity by Judge Murray, July 3d, 1888. History obtained from his sister. Patient is the youngest child of a family of four children. His father was some twenty years older than his mother, being fifty-six years of age when he died. His father had been suffering from consumption for some years, and on several occasions before patient's birth was thought to be dying. He only lived a few months after birth of patient. On account of the illness of the father, the mother was anxious during the pregnancy with this child, and often was deprived of sleep and severely taxed physically by the care and nursing of a consumptive husband. The patient was a weak and sickly baby and until after the age of two years was not expected to survive. He received a fair school education but never displayed any force of character. During his school days he made but few friends. The only intimate friend he ever had became insane some years ago and is now in Bloomingdale Asylum. On account of his delicate health he has been petted and indulged. He has never

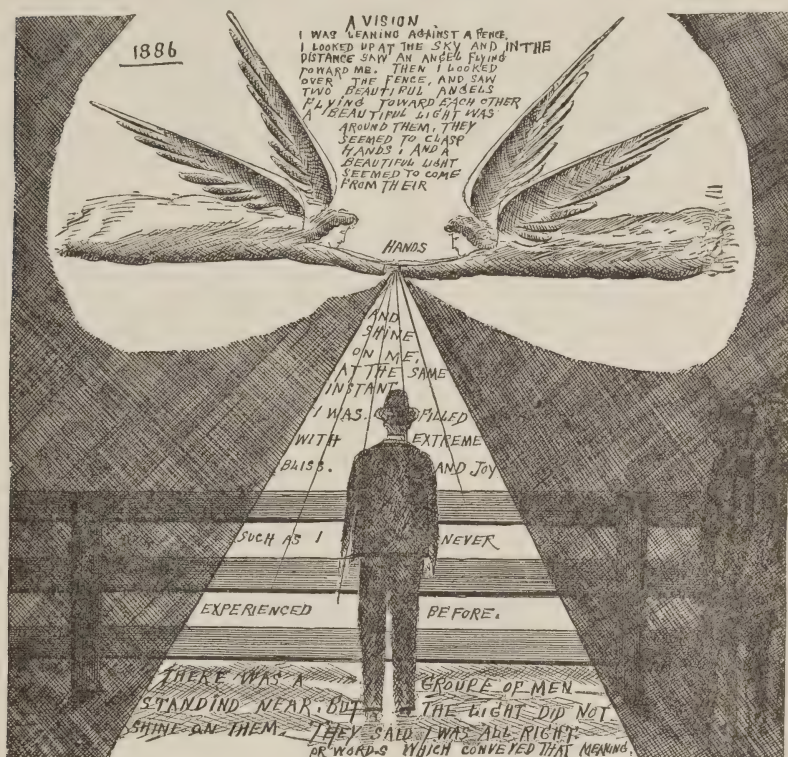
¹ Read before the Neurological Section of the Academy of Medicine, 1889.

been able to support himself. From boyhood he has been solitary, moody and irritable ; having outbursts of passion from slight causes. Shortly after obtaining his majority he went west where he remained for about eighteen months. His family know little of his life during this time. He came home bringing some photographic apparatus and said that he had acquired that art while away, and had ambition to become an artist and devoted much of his time to drawing. He claimed to have been victimized and cheated while away. He now became more solitary than ever and had no associate outside of his family and made no confidant of anyone. Occasionally he was sociable with one sister but never confidential. He has kept to his own room and his family left him to himself and never disturbed anything about his room. He did a little work in the way of "touching up" photographs. He always brought this work home. For the past two or three years he has been more moody, irritable, and at times would harangue and become much excited. His theme was usually religion. Then his sister discovered curious pictures of his own production hung about his room. He then began to talk to his sister about the star of Bethlehem, and evenings she would find him gazing at the sky and he would point out a star, saying, "that is the star of Bethlehem, that is my star." Then he told her of visions that he had seen, and that his drawings represented these visions. Then he refused to take food for a time and said it was not necessary for him to eat as God fed him. He now began to talk more strangely and was even more solitary. They learned that he had been arrested for creating a disturbance upon the street. His sister assured me that he had always been an inoffensive fellow and had never before interfered with anybody, and although he had asserted that he was ordained to perform certain things for the good of the world.

Examination : He has a weak face and a silly and pompous manner. Poor physique. Narrow and highly arched palate.

He stated that he had been arrested for preaching on the street, and attempting to stop the cars. He did not

directly interfere with the cars, but shouted to the drivers that they were breaking the Sabbath and that the wrath of God would descend upon them and that he had been commanded to warn and expostulate with them. He then said



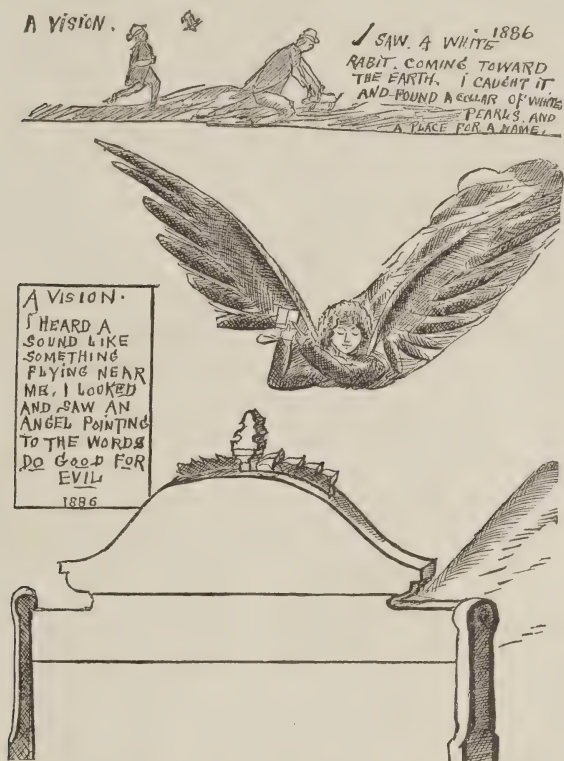
that in 1886 he saw three visions, and that in these visions it was revealed to him that he was to reform the world. These revelations came from God ; that he saw Jesus Christ pointing to him and he felt that he was raised above all evil and above all of the devil's work. These three visions came to him early in 1886 and had never been repeated. He was much confused in his descriptions, but said that he would represent them by drawings. He made two sketches with a pen which I will pass around. He made another drawing which he gave me the following day with written explanations. He writes in the upper corner, " These vis-

ions happened in the year 1886." Then below the first drawing he writes, "A vision. I seemed to be standing against a fence, I looked in the distance and saw an angel flying toward me. Then I looked up before me and saw two beautiful angels flying towards each other. A beautiful



light was around them, they seemed to clasp hands, and a light seemed to come from their hands and shine on me, at the same instant I was filled with extreme bliss and joy, such as I never experienced before, the group of men standing in the distance seemed to remark, 'he is all right' or words which conveyed that meaning, the light did not shine on them." The second drawing is very crude and like the first is an effort to represent what he describes, he writes in the corner: "A vision. I seemed to be lifted up by our Saviour in great triumph and all the buildings were falling with a great noise." The third drawing is given with this description: "A vision. I looked up and saw a snow white rabbit coming toward the earth. I ran and caught it and

found a collar of white pearls and a place for a name. A vision. I heard a sound like the rustling of the wind, I looked up and saw a beautiful angel pointing to the words 'do good for evil.'"



The patient was taken home by his brother and sister who wished to care for him. He told me that he had finished drawings of these visions done with india-ink which he would give me if I wished. But he never sent them to me. A few months afterwards I went to his house to try and get these drawings. He heard my voice from his room and refused to see me, and when told by his sister that I wanted to see him he became much excited, and in very emphatic language announced that he would not see me or give me any drawings. It was evident from his language that if he had been raised above all evil, there was no evil

in profanity. His sister informed me that for a long time after his arrest he would not leave the house at all, and then made her go with him when he went out, it was only after some months that he ventured out alone. He complained that all of the clergy for whom he had done everything, abandoned him when he was persecuted by the world. The sister also states that he is never threatening in his actions or words but frequently delivers excited and incoherent harangues, generally about religion and the failure of the world to appreciate him.

During September 1889, he was again committed for examination, having become excited at a relative's house.

His mental condition had changed but little. The same delusions existed and the connection with the three visions were just as apparent. For the first two days he was very quiet, and was not inclined to talk as freely as upon the former occasion; evidently fearing that he might be sent to the asylum. The third day he was very talkative and discoursed freely about the visions and his delusions. Finally he became very much excited and declared that he was the second Christ, and that all who neglected him neglected God and the Holy Spirit.

This has seemed to me to be an interesting case of systematized delusions occurring in a feeble minded individual; where the delusions followed and were built upon visual hallucinations that have never been repeated, though three and a half years have elapsed.

It is my experience that visual hallucinations are rare in cases of paranoia and it is still more rare to find them the standing point of the delusions yet not repeated. It is more usual to find auditory hallucinations and to have them continue, and to govern a prominent factor in the delusions.

I, however, recall one other case, that of Arthur D. Austin, who attracted considerable attention in this city a few years ago. In his case there was a single vision that was not repeated. He could give not only the date, but the hour and minute of its appearance, and its actual duration. From the moment of this single vision he was an altered man.

CASE II.—M. L., aged 56, single, domestic.

No family history. On admission she was in a filthy and neglected condition. She resisted interference. She told me "My husband is an officer in the upper land." "He is the Heavenly Father's son." "He first presented himself to me on Oct. 1st, 1868." She did not see him when he first presented himself but she was told by God's voice that on the next Sunday he would sit beside her in the pew in church. The next Sunday, Oct. 4th, 1868, she saw him in the seat with her and she describes his personal appearance. As she was leaving the church she heard some one remark, "she does not know her husband." She says that she knows that she has many sons and daughters in heaven though she has never seen them and knows that they were not born in this world. She has been told this by God's voice. She has been hearing the Father's and the Son's voices ever since Oct. 1868. She has been and is still waiting for her husband to come with a procession and carry her away as he has promised her to do.

At times she is quite coherent and speaks of her Father's being the heavenly Father's grandfather, etc. In this manner, and by her indifference to her surroundings and neglect of her person she displays much dementia. But if we consider the duration of the delusions (*i. e.*, twenty years) the dementia is slight.

In this case the hallucinations were auditory, and were the foundations of the delusions and continued to form a very important feature of the disease. The delusions continued unchanged for over twenty years and she is at this time able to give the exact date of the first appearance of the hallucinations.

CASE III.—S. D., aged 38, married, inventor.

Family History.—Paternal side good. Father intemperate, but a man of education and marked ability. Maternal side defective. Mother consumptive. Grandfather insane and grand-uncle insane. Patient is the youngest of four children. The elder sister is a rounded character in everything, educated, refined, and practical. Next, a brother, has been odd, a good thinker, but from his family connec-

tions noticeably weak in character and lacking in ability and ambition. This brother's children are remarkably bright and capable. The next, a sister, is certainly peculiar, while kind-hearted, is a gossip and a great disturber in the community where she lives. She makes much of little matters, and is very erratic in actions and conversation. At times is very bright and entertaining.

The patient is a man of magnificent physique, six feet and two inches tall, and weighs from two hundred to two hundred and twenty pounds. Has displayed marked ability from early childhood. He was considered a genius ; I may add that he is the only person that I ever saw who seemed entitled to this distinction. He was eccentric in some ways and always a poor business man. His inventions have given him a world-wide reputation. It was always difficult to get him to perfect his inventions, as, after demonstrating the principles, his interest would diminish. He was an erratic worker, becoming all-absorbed in some problem, and, when he got the idea down on paper in the shape of drawings, he would throw everything aside, and was ready for anything that was going on.

The manager of a large company where he was employed said his knowledge appeared to be intuitive, as he would enter the operating-room, where the instruments were generally giving trouble, and that he would pass from table to table, and at his first touch of the instrument would find the exact fault and quickly remedy the defect. In a short time everything would be in order.

Some four years ago he undertook the management of a company where he had much executive and business work to do as well as the exercise of his own profession. This work greatly harassed him, and he became very irregular in his mode of living, sleep was interrupted, and he drank freely, though rarely, if ever, intoxicated. He became much run down physically, and it was thought best that he take a rest. He was started for Europe with his wife early in December, 1885. Almost as soon as the steamer left the dock he exhibited delusions of persecution. Was suspicious of everybody, declared that there was a conspiracy to de-

stroy him. He attributed every action of his fellow-passengers as relating to himself. When several would be conversing anywhere, he would feel sure that they were talking about him. He found that the ship was taking the southern and longer route, and asserted that this was part of the conspiracy to get him farther from land, and, when they would be at the one point most distant from all land, throw him overboard. He believed that certain passengers were employed by a Californian, with whom he had had trouble a number of years before, to follow him and make way with him. This man was a Jew, and he became especially suspicious of all Jews. Then he saw certain signs that were made by these people, and he said they were Masonic signs, and next he had all Masons with the machinery of that secret order working against him. He was a Mason himself in good standing. This state of affairs continued, but kept getting worse all the time. He continued to travel, and every new sight and face suggested some new idea. After a few weeks his wife induced him to return, which he was glad to do. His delusions became so active on the return voyage as to attract the notice of the other passengers. He was threatening toward one person, and his wife was in constant dread of some encounter.

I saw him almost immediately after his arrival, and found him almost completely given up to the contemplations of these delusions. He would not move about without scrutinizing everything. In leaving or entering a house, he would look about. If there was a screen or portière in the room, he would not be contented until he had inspected what was behind them. He now commenced to have unlucky numbers, and he would by almost lightning calculation make out an unlucky number from every combination that he saw. He now suspected all organized bodies as being in the conspiracy. He was more bitter against his late business associates than any others, and would give the most trivial reasons for his suspicions. Yet he always had reasons for all his beliefs, and would stoutly defend his ideas. He said to me one day that he spent his time dove-tailing everything that transpired into these ideas of his, and this was

literally true. When confined to his own house, where everything was familiar, and when no one came to see him, he was quite calm and free from these ideas, was pleasant and companionable, and ate and slept well. One attempt was made to send him to a private retreat without certificates, but in twenty-four hours he left the place. While there he did not sleep, but sat up all night watching the door and windows, which he had barricaded, and he refused all food, fearing that he was to be poisoned.

As soon as he arrived at this town he saw a poster giving notice of a meeting of the Grand Army of the Republic in an adjoining place. This meeting was for him, and he had been entrapped. The physician's name was Mount and a servant's name Moria. Here it was again Mount Moria, and this was the name of his Masonic lodge. These two incidents will illustrate fairly the thousands of explanations he was giving to every trivial incident, and how he was forcing his own personal identity into the actions of everybody he chanced to meet. Every look and movement of people he met, every notice and poster, was translated as having a bearing upon him.

One day he appeared at my office and wanted me to look at the roof of his mouth, which I did, and discovered a small sore, the nature of which I explained to him ; but he declared that it was a chancre and he knew just how he got it. While on board ship, he said, he observed that they always gave him his cigar out of a particular box, and that these cigars, infected with syphilis, had been prepared for him. He had always been suspicious of those cigars, but he had not been able to divine their designs until now. The explanation of this delusion was this : he had seen a copy of the *Medical Record* that contained an editorial on the lewd pictures of cigarette-girls that were then conspicuously displayed in all cigar-stores, and remarked that girls who would allow themselves to be photographed in such positions would be likely to acquire venereal disease, and added that it is well known that syphilis has been conveyed in cigars. This was sufficient to convince him that the sore in his mouth was a chancre.

Finding that he could eat and sleep well in his own house, and was there more quiet, and to avoid his meeting new people and seeing new sights, I found a young man who was willing to work with him and act as a companion and who would keep a constant watch over him. I then had him begin his work, a shop being built, where he continued his inventions and made his own models. He began to improve, and has, with remissions, continued to improve up to the present day. He now manifests but slightly any of these ideas. I remember one night he was at a dinner, and was really the life of the party; during the afternoon of that day he had told me for an hour of a conspiracy to ruin him; he was then free from the fear of personal injury.

He is still suspicious and superstitious, and maintains a belief in some of the delusions. Every once in a while he will show evidences of these delusions, and his conduct will be guided by them.

I have had opportunities to observe the progress of this case, and no sensory hallucinations have ever been manifested.

During the past three years he has done some good work, but whenever he attempts to have business transactions he develops delusions of persecution. He has never had delusions of grandeur.

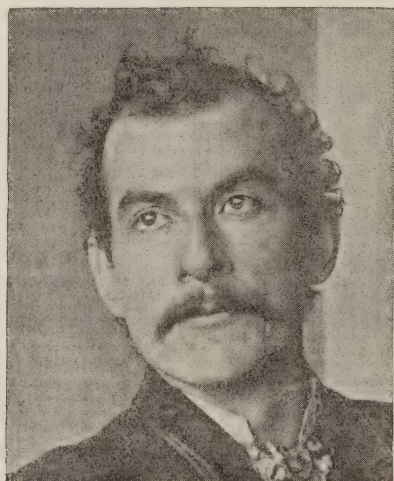
CASE IV.—J. M. D., aged 33, single, canvasser.

No family history except from patient. From his account there is no heredity. He thinks that he has always been considered eccentric. A lineman who knew him some three or four years ago, stated that D—— then talked much of Miss Anderson, and believed that she as well as many other ladies above his station in life were in love with him, and were constantly making overtures to him. He was suspicious, egotistical, and would think that the actions and conversations of others related to himself. His fellow workmen would take advantage of this and tell him all sorts of stories; he seemed to have a fear of detectives, and they would sometimes say that detectives were after him, when he would leave his work and clear out. His own ac-

count of his life is, that he received a common school education and when a mere boy started out to care for himself. For some time he worked in a factory, finally went West, and was a prospector and a miner. In the mines he made a few thousand dollars, but asserts that he was cheated out of a fortune. Then he was a telegraph operator, and then a lineman, and lately has been canvassing for Appleton's Encyclopedia. For years he has been trying to study and explain all natural phenomena and is writing a work to elucidate all these subjects. The creation of the world, geology, astronomy, chemistry, etc., will be explained and harmonized. He believes that he has been very attractive to ladies, that they have followed him about in disguise. He assured me that he could have married ladies of rank and fortune. After Cleveland's election, four years ago, he sent him a long congratulatory telegram and volunteered his services. Some six years ago he first saw Miss Anderson, and fell in love with her, as she did with him. When he was in the audience she always played to him, not to the others. Every look and gesture was addressed to him. He wrote frequently to her and sent her photographs of himself and locks of his hair. He found a photograph of Miss Anderson taken as representing Rosalind, and he said the hat and dress was what he used to wear in the mines, and this had been taken for him to show him her love for him. He followed her about from place to place, two or three times to Europe. She followed him in the streets, being disguised, but he knew who she was. He never tried to speak to her but once. This was when she returned from Europe last September or October. He procured a pass for the revenue boat by representing himself as her fiancée. On board the steamer he spoke to her but was not offensive and attempted no liberties. He followed her about to be near her as a protector. Her every action and the action of those about her he conceived to have reference to himself. He saw in her actions and professional engagements constant reference to himself, and was waiting for the one word that was to bring him to her side. He saw that all associates and admirers knew of him and that he was the

avored suitor. Last summer he visited England and here he developed delusions of persecution. His rivals for Miss Anderson's affections saw how he was favored and they conspired to put him out of the way. At Liverpool he was poisoned in food at a railroad restaurant and he nearly died. Shortly after this he left England for home expecting to die from poison. Before leaving he wrote Miss Anderson of his condition and plans. He came to Washington and began to canvass again. He believes that she followed him to Washington, when she was supposed to be in a convent in England. She could not remain away from him when he was ill and suffering. When she found he was able to be about she did not announce herself to him but took rooms in a house directly opposite to where he was lodging. He could hear Miss Anderson and others talking about him. He is possessed of such remarkable hearing that he could distinguish their voices and conversation that distance. They had mirrors arranged in such a manner that they could watch his movements in his rooms. He evidently now began to have auditory hallucinations. He frequently saw Miss Anderson, he says, but I questioned if he had any distinct visual hallucinations; there was evidently some real person whom he saw. These persons he believed to be Miss Anderson in disguise. When Miss Anderson began her professional engagement in this city, D—— put in an appearance and believing that his one great mistake had been that he had held back from declaring his love and that she was angry with him on this account, began again to write letters to her. He visited her hotel and sent letters to her rooms, and stated that he was waiting for answers and would remain for hours about the hotel. He told of his persecutions, and that he was prepared to defend himself. On the 14th of November he was arrested, and the following day I saw him at the pavilion for the insane at Bellevue Hospital. While at the pavilion he had an air of importance, and read every newspaper allusion to himself and was much puffed up by the space devoted to him. He wrote a long statement for publication which he gave me and desired that it should appear just as he had written it.

Portions of this were printed. He was very angry with me that such a garbled account should appear, as he had instructed me particularly that he wished to correct the proof himself. He informed Dr. Peterson, while I was conversing with another patient, that he was sure that I was in the conspiracy against him. When arrested he was armed with a self cocking revolver with five chambers loaded ; each bullet would weigh a trifle less than half an ounce. There was also found in his pocket a sheet of letter paper ; at the



top of this in large letters and underscored was the word "Notice" and below was written, "Sometime I may be found dead, or in a trance. Ifso, you can safely say I have met with foul play at the hands of Abbey and that gang. P. S. No matter where my body may be found, ship it to Miss Anderson. She will see that it is interred and attend to all the funeral arrangements."

I will now read some abstracts from his own statement of his case. I will also pass around this photograph of the patient and a tracing of his head made by Dr. Peterson.



The pathological variation is in βX and βB , and hence although below the average in most of its measurements, the head of this paranoiac is above the average in the naso-occipital arc and binauricular arc, owing to the pathological height of the skull.*

	Average of normal male skull	Limits of physiologi- cal variat'n	Jas. Dougherty.		Remarks.
			Head.	Skull.	
Circumference.....	52	48.5—57.4	54.7	51.5	Below average.
Volume (rough approximation)	1500	1201—1751		1390	Below average.
Naso-occipital arc.....	32	28—38	36.5	34.3	Above average.
Naso-bregmatic arc.....	12.5	10.9—14.9	12.5	12.5	Average.
Bregmato-lambdoid arc.....	12.5	9.1—14.4	12	12	Below average.
Binauricular arc.....	32	28.4—35	37	34.7	Above average.
Antero-posterior diameter.....	17.7	16.5—19	18.8	17.8	About average.
Greatest transverse diameter.....	14.6	13—16.5	14.8	13.8	Below average.
Length-breadth index.....	82.2	76.1—87	78.7	77.4	
	Brachy- cephalic		Mesali- cephalic	Dolico- cephalic	Below average.
Binauricular diameter.....	12.4	10.9—13.9	13.2	12.5	About average.
βX (Bregma to X).....	11.7	10—12.65	13.4	12.9	Pathological.
Facial length.....	12.37	10.5—14.4	12.5	12.5	Above average.
Empirical greatest height (βB)	13.3	11.5—15	15.5	15.5	Pathological.

* PREPARED BY FREDERICK PETERSON, M. D.

PIQUED.

Jimmie and Mamie.

Mary Anderson Visits this Country on the Sly to see
Dougherty.

Rosalind on the Rampage.

After her Jimmie, while supposed to be in a Convent in
London.

“Passing over the earliest portions of our love affair,
which tho' interesting to us might weary the reader, I will
that which will explain the motives of our recent actions.

While in Liverpool on my last trip to England, I was severely poisoned in a glass of liquor in the 2nd. class refreshment room of Northwestern R. R., Line St. Station, its effects were very severe but I managed to cure it well enough to take care of myself. The impaired stomach caused by this drugging run me down so low in vitality that I fear'd that I would not live to see this November. So I wrote to Miss Anderson bidding her a good-bye, telling her I probably would not live to see her again. This was some time about the latter part of July last. About the middle of August I attended St. Patrick's Church in Washington to vespers, as I came out after services a woman's voice sounded "Oh! What can I do when he wont speak to me." I may here state that from the first Miss Anderson refused to be formally introduced to me or answer my letters. But seemed anxious to form my acquaintance in a more romantic way. * * *

I now felt certain of her, but made up my mind to let her run awhile as she did me in London. That evening I was sitting in my room, when I heard the gruff voice of a man outside the door of the house opposite say, "You can't ever make anything out of that dude, let 'em go." A voice like Miss Anderson's answered, "Cheese it; he's onto us watching him." She evidently was standing with him as he uttered his advice, and she spoke her reply as she dodged into the house, I at the time turning to look at them. It was quite dark, so that I could not recognize what the man was like, but from his voice believed he was a short, thick-set, florid, "duffer-like" man, such as I had seen with her afterwards in the Temple Café.

She then evidently took a trip away for a few days, for I heard the same young woman's voice which did the bantering in the carriage say the following evening, "I don't know what she wanted to go for. She's just as crazy after him as she can be," adding, "of course she is," to a grunt from an elderly man, her companion. This man then in an irritable old honest farmer-like voice said: "These actresses are a poor lot. They're frauds, all of them; not only that but they're mean," following it up with a tirade against acting in general.

The next I heard of her was three or four evenings afterwards, while sitting in my room, picking up her picture, gazing at it for a time, finally ending by kissing it. As I did so there was a couple of little jesting laughs from two females in the house opposite, and "Ha, my picture! Maybe he won't like me when he sees me," in a sort of happy, uncertain, subdued voice from our Mary, who evidently was sitting with them watching me.

The next day I kept a close watch upon the house. During the day a carriage drove up and a lady alighted and entered the house. I concluded 't was probably some visiting friend of Mary's, and that now was a good chance to hear a confirmation of my former opinion at close range, so I left the house with a book in my hand, as usual crossing toward F Street, so as to pass near the house. The sitting-room windows were open but covered with lace curtains. I was amply rewarded. They had evidently observed me, and as I approached the visiting lady said: "It's too awful bad. He's just grieving himself to death after you," and Mary responded in a subdued voice that seemed as though she was speaking guardedly and watching me at the same time, "Yes, after my picture, but I don't believe he knows me." I was within a yard of the open window and within ten feet of her now. I felt like catching the curtain and pulling it aside, but concluded to let her run and fix the meeting to suit herself, as she refused to accede to my way. Now confident that 't was she, and worrying to meet her, I changed my mind that afternoon, went to the house and asked if she or the other strange lady were stopping there, but was answered "No. They had n't even seen in the papers that Miss Anderson had returned to this country."

I then went to the Sisters' Orphan School near by and asked one of them to undertake the task of arranging a meeting between us, but the sister only looked incredulous, and said it was worse than a fairy tale, and said if Miss Anderson had come to Washington it would surely be in the papers. I tried to explain to her that under the circumstances it surely would not be in the papers, and that I

would pay her for her trouble. I could see that the utter absurdity of Mary Anderson's coming after a man that looked as I did then was too much to believe, and all the urging I could do in the matter could n't induce her to act.

The next of "Lovely Mary" I heard was that evening while observing the planets Jupiter and Mars. To a question asked by some companion she replied, "Yes; he knows all about them. One of the first things he ever asked me was to throw my horoscope," I may here state that I never asked that privilege of any woman except "Mary."

That night, while worrying over how to get matters arranged, and filled with an extra dose of lovely excitement through thinking of her so continually, I was worse sick than usual, and was forced to rise during the night a number of times for the purpose of taking hot drinks. She and the others were evidently on the watch, and she remarked: "Do you think he was poisoned?" Her companions, scouting the idea, replied: "He's in love; its love," she retorting: "He may have been poisoned and in love to." I may here state again that I have told no woman of my poisoning up to this time except Mary. To make matters still worse, my head ached, from indigestion, I suppose. While having my hand on my head nursing it, Mary uttered in a broken voice: "Oh, God, do I deserve this?" Either her intercession for me or the knowledge of her solicitude helped me, for my head got over the pain at once.

The next morning as I was returning from the office she, as usual, was on the watch, and remarked in an intensely eager sort of fidgety voice: "Jesus, don't I wish I was that man!" This, coming from my idol, kind of startled me, and I made up my mind to let her run and learn her traits of character on the sly, as she evidently supposed she was doing with me. Afterwards, as I was brushing my hair, she said to this companion: "He is awful proud of his hair; he sent some to me a couple of times." I may here add that I never sent my hair to any woman except Mary, and that I sent in two letters, leaving a chance for one to not reach her. * * *

The following Sunday morning I heard my Mary say in an introspective, wearied voice: "Do you think he wants to marry me?" to which the companion burst out the reply, as if Mary's too ludicrous position and question caused her mirth, "Of course he does." * * *

Next I heard from her was: "If I knew as much before as I do now I would have been married years ago." This she said that evening in a meditating, retrospective tone of voice. A couple of days after she said: "He don't look a bit like he used to. I don't even like him now. Oh, he used to look grand!" * * *

The last I heard of her she said: "I'll speak to him anyway, and if he loves me I'll take him home with me." I thought I had conquered, but she evidently changed her mind, or it may be that the opportunity did not occur and that she was forced to go in a hurry to attend to business or catch the ship to return and fill her provincial engagements in England, and upon sober thought, coupled with the urgent advice of friends, concluded that, as I was broken, the best thing she could do was to give me up, or she may be suffering from a bit of pique at my seeming intentional slights. I meant the most honorable in the world toward Mary, but sought to break her terrible self-will. I have broken myself instead, while Mary sails on as of yore as an "Ajax defying the Lightning." * * * After this terrible exposé I feel that all must ever be over between us, and fully realize my great loss. She has trivial vices that cling to her from her surroundings as a smoky odor. To the one who works among it; but the mind of the woman is as far above her surroundings as heaven, purgatory. The current belief that Mary is a niece of Marble is all wrong. She appears cold as the cloud-crested volcano to the verdant grassy hillock, so is Mary compared to other women. She has possessions as herself so far above the ordinary that the ordinary cannot understand. They watch and like, but don't understand, passion; she is filled with passions and riven unknown, but the surges of the woman's heart can never do more than gurgle through and soften her nature. The wondrous mind is ever in control.

I shall feel my loss, and believe Mary will remember"

JIMMIE

DOUGHERTY.

"If the mere fact of my running after my sweetheart without the formality of an introduction constituted a sufficient reason for my being considered insane, I am satisfied that more than half of the now married men have deserved this punishment at some period of their lives; also if such were the case, Mary, as I show above, should be sent to the Island in the same boat with myself. If, however, she is suffering from a fit of pique over my supposed slights, in justice the statements here made should be looked into, and if found true, I can only see that I have erred more in omission than commission. That she was recognized by many during her stay in Washington I feel assured.

* * * If, however, all that I have stated are conjured phantoms of a diseased brain, then I am only too thankful that I live in a country where such good institutions are provided for my welfare, and shall only be happy to avail myself of the privilege of entering such. If my brain is really affected, it is probably due to excessive study in trying to raise myself to a position that would enable me to make my advances towards my Mary from at least a somewhat equal station, as during the last five years I have been furthering a set of discoveries that will mark an epoch in the advance of natural science, in a book which I am now preparing. I will give a theory on the probable forms of atoms, an hypothesis on the structure and action of molecules, together with an explanation of the cause of molecular motion. In it I will give a history of the creation of the earth, that will accord with both history and geology, and account for its various phenomena as they now exist. In it I will show the laws which govern meteorology, why the clouds float above an atmosphere much lighter than themselves, sometimes giving out their contents and at others not, also a possibility of governing storms, at least to a certain extent. I will fully explain the cause and action of the sun's external light and heat, and give further testimony in support of Keppler's "*Harmonies of the World*;" will prove that evolution is a necessary consequence of the natural law which rules the universe, coupled with the "*kaleidoscopic*" configurations of its portion. I will also prove that

there is some truth in astrology and "Ptolemaic theory"—in fact, I find that nearly if not all of those old exploded views that swayed the world in times past had something in them. I have proved "Avogadro's" law wrong, and Newton's theory of gravitation as being only half right, and if I must pass on to oblivion through insanity I will at least leave my mark upon the age I lived. It is a necessary something through which all must pass in some way and time. 'Tis but a deferred respited sentence at most. My manuscript as it now exists in its unfinished state will lead the casual investigator to but more thoroughly believe in my insanity. I humbly ask that it may be saved and given to me if I be put in the asylum, that I may finish it before my brain is totally gone. My private letters from my friends and relatives have been destroyed as "trash." They may appear so to the authorities, but they were not trash to me.

If my investigators will but give me the benefit of the doubt till proven insane, if they will look through history and find examples numerous where men, not understood, were termed "cranks," subjected to various tortures, although they helped posterity, which now honor them with monuments; for my own part I would much sooner have the grasp of a friendly hand now than a million dollar monument by posterity."

J. M. D.,

Bellevue Hospital, Nov. 17th, '88.

The delusions of grandeur were developed slowly and have continued for years with little change. Delusions of persecutions have appeared, but they have developed from those of grandeur, and are in every way conditional upon them.

The late appearance of the hallucinations is rather unusual. The exaggeration of the ego displayed throughout is very characteristic of paranoia.

This case is not unlike Esquirol's Esotomania, a subdivision of Monomania according to Esquirol. "This perversion is not necessarily accompanied by animal sexual desire, and the adjective *crotic* is here used in the classical

sense. The patient, noted in his adolescence for his romantic tendencies, construes an ideal of the other sex in his day-dreams, and, subsequently, discovers the incorporation of this ideal in some actual or imaginary personage, usually in a more exalted social circle than his own. He then spins out a perfect romance with the adored personage as its subject, and, according to the external circumstances, appear to him momentarily favorable or unfavorable, expansive or depressive. Delusions are added to, incorporated with the crotic ones.

As a rule, the affection for the adored object remains as chaste and pure as it begins — a sort of distant, romantic worship; insane for the reason that unimportant occurrences, accidental resemblances, facts which have no natural connection with the individual, or his or her real or imaginary contemplated partner and hallucinations, are woven into the delusive conception which, consequently, assume such a predominating position in the patient's mental horizon as to entirely overshadow it." SPITZKA.

Case No. V. Hugh Fraser, age 58, married, clerk. This patient came to Bellevue Hospital on May 4th, 1888, and asked to be sent to the Island. He was much agitated and declared that he had syphilis, and had infected his family and visitors to his house; that two-guests of his son had been infected by him and had cerebro-spinal meningitis. He declares that "he has lost all his manhood, and that he has the blot upon the brain." He will not use a comb belonging to the Ward, as he says "some one might become affected." He begs to be sent to the asylum, as he is sure that his sons will murder him when they find out how he has wronged them. They do not want to injure him, but when they know it all they will not be able to restrain themselves. His sons took him and tried to care for him.

On May 22d, 1888, he again came to the hospital and begged to be sent to the asylum. He was somewhat excited and threatened suicide. He expressed all the delusions he had on the first occasion; declared that he was wasting away and that his feet were like dough; that he

had infected his family and was a monster and deserved death, etc. He was again taken home by his sons.

On July 7th, 1888, I was conversing with the house physician in front of the pavilion, when we saw this man come into the hospital yard. He was trotting along, and went to the office and then to the eastern end of the pavilion, and then to where we were standing. He was very agitated and begged to be admitted. When objection was made to this he fell upon his knees and fairly supplicated and beseeched to be taken in. He declared that he would be murdered if not protected, as his children were finding out how he had infected them, and repeated the old story. When it was decided to admit him he seemed greatly relieved and quieted down. He told how he had escaped from his own house by climbing out through the scuttle and then from roof to roof until he reached a tenement house, where he made his way to the street. He thinks that his mouth is full of dust, and does not want to eat, as nothing can pass his bowels. He is sure that he is bound to die, as the syphilis is consuming him. He believes that his sons have general paralysis, due from infection from him.

He probably has had syphilis, but at this time presented no physical evidence of the disease. I mean no evidence of activity. He was sent to the State Asylum at Middletown, N. Y. Under the date of November 26th, 1888, Dr. Talcott writes me: "Mr. Hugh F—— has not changed very much in the character of his delusions until within the past two or three weeks, when he became more irritable and suspicious, thinking that electricity was connected with his bed and that his people were outside the ward anxious for him to come out and go home. On speaking to him he becomes very much excited and abusive. He thinks my assistant is conspiring to keep him here against the wishes of his friends. This is all that we can get from him at present. He is eating and sleeping fairly well."

The delusions in this case are systematized and have existed for a long time, certainly six months almost unchanged in character. Though hypochondriacal in char-

acter, they are logically reasoned out from his false conceptions of syphilis, and upon this all his delusions are founded. The actions of his family he attributed to their knowledge of his infection of them, and their illnesses, and those of people who had visited his house he believed were the results from contamination from him, and in view of these, to him facts, he was agitated and depressed. He has developed no sensory hallucinations.

From Dr. Tallcott's letter it would appear that the delusions were changing in character to those of persecution.

Morel, in 1852, gives some observations on the transformation, among the hereditary insane, of hypochondriacal ideas into ideas of persecution and afterward of grandeur.

Kraaft-Ebing distinguishes two kinds of paranoia.

First: That with delirium of persecution, which he fully describes with its three stages of hypochondria, of persecution, and of grandeur and its sub-variety "quarrelling insanity."

It will be interesting to learn if this patient develops ideas of grandeur in the future.

I do not wish to be understood as advocating the classification of this case under the name paranoia, as I think the term is being made too comprehensive by some. The man's age is certainly against paranoia. I regret that I am unable to give the family history and the story of his early life.

Editorial.

ELECTRICAL RESUSCITATION.

The late Dr. Griswold, just prior to his death, made some very exhaustive and valuable experiments upon dogs, with the view to find out just what action galvanism had upon the heart and lungs, and whether or not it was depressant in character applied directly or indirectly through nerves in region of the neck. His experiments and their practical results he published in the *New York Medical Journal*, April 4, 1885.

The subject was briefly though thoroughly investigated, and all his experiments verified by us, and the practical *résumé* was, that galvanism was a depressant, and dangerously so when applied in cases of collapse or shock. In dogs, when either aconite or chloroform narcosis was present, and a moderate current used, one pole over the apex and the other over the course of the pneumogastric and phrenic in neck, the heart ceased beating instantly, and respiration ceased as well. In our teachings in the past we have endeavored to impress the fact that galvanism is dangerous, and ought never to be used in resuscitation of persons who have ceased breathing or have sustained heart-failure. The strong faradic current, however, as a counter-irritant and as a stimulant through reflex action, applied to nipple directly over the heart or by puncture to some nerve-trunk, was of decided benefit. We are glad to note that this is sustained, and especially Dr. Griswold's experiments verified by Drs. H. A. Hare and Edward Martin, in the Warren Triennial Prize Paper, of the Massachusetts General Hospital, entitled "The Value of the Electrical Methods employed for the Resuscitation of Persons who have ceased Breathing" (*University Medical Magazine*, vol. ii., part 2), the resume of which is :

"The action (of galvanism), under these circumstances, on the heart was most striking, for it was found that the only place where the positive pole could contract the diaphragm *also inhibited the heart*.

If a current, by no means as strong as that frequently used in cases of suspended animation, produces such a profound effect upon the heart of a moderately anæsthetized dog, its effect upon a heart already overburdened by a congestion or depression would be disastrous.

It would seem probable that in those cases where the use of electricity has been resorted to, the return to life has been the result of reflex stimulations rather than a direct effect on the phrenic nerves. On the contrary, the striking effect upon the heart, shown by tracings, suggests the thought that improper application of electricity may in the past have been an important factor in determining a fatal issue."

"The observations of these gentlemen fully confirm those of Dr. Griswold, and are fully endorsed by us.

We regret exceedingly that Dr. Griswold's most excellent paper had not been known to these gentlemen in helping them to quick decision as to the dangerous effects of galvanism in cases of suspended animation.

THE PROPOSED NEW LUNACY LAW FOR NEW YORK STATE.

It will be remembered that, at the last meeting of the New York legislature, a lunacy bill was introduced which contained a number of sweeping reforms. The bill originated in a committee of the National Conference of Charities, and was engineered through the legislature by Mr. Gallup. It failed, however, to become law through the neglect of the Governor to affix his signature.

The same bill—with, we believe, no modification—is to be reintroduced at the coming session; and we desire, therefore, to call the attention of alienists in particular to some of the features which embody great improvements or impor-

tant changes. We may briefly summarize its most admirable characteristics as follows :

Removal to Asylum.—Attendants of the same sex must accompany patients to the asylum.

Voluntary Patients.—An insane person, cognizant of his condition, may at his own pleasure enter an asylum by written application, accompanied by a certificate from his family physician. He can leave by giving six days' notice to the asylum officials and two days to his family physician.

Emergency Cases.—Dangerous or exhausted patients may be received at the asylum for three days without procedure of any kind.

Confinement of Insane in Jails.—It is forbidden to detain a patient in jail beyond ten days, or to confine him in the same room with criminals.

Home Furloughs.—Visits home by asylum patients, of indefinite duration and at the discretion of the superintendent, are permitted.

Boarding-out of Chronic Insane.—This system, practised successfully in Scotland and of late in Massachusetts, is provided for in the new bill.

All of the features enumerated are important for the welfare of the insane, and our present laws are sadly derelict in respect to these matters.

For instance, to illustrate the faults of existing laws in the points just mentioned, we need but call attention to the fact that insane women are now, as a rule, taken to institutions by male deputized officers, who are not infrequently intoxicated ; no patient can go voluntarily to an asylum without recourse to the usual two certificates, approved by a judge ; it is illegal to admit a patient without the two medical certificates, even if such patient be moribund or in great danger of doing harm to himself or others ; it is not uncommon to detain insane persons in jails, with criminals for company, for indefinite periods of time, in country districts ; no home furloughs are at all permissible except by evasion of present laws ; the boarding-out system is of course novel in America, and has not been provided for as yet in this State.

From all this it is easy to see how many exceptionally good qualities there are in the bill.

Its one great fault lies, however, in the method of commitment it presents. This is pernicious in the extreme. The manner of admission to asylums, as now practised, seems to offer obstacles enough to the early hospital treatment of these unfortunates; but the new bill quadruples these obstacles. The procedure proposed is so complicated, requiring no less than thirteen papers, and so surrounded with legal formalities, is so open to abuses of a political nature, that it should not be permitted to become law. As has been well said, an insane man is not a criminal, but an invalid requiring hospital care.

But our readers may draw their own conclusions as to the advisability of changing the existing process for one more difficult and confusing, by glancing over the steps in the method of commitment proposed, which may be summarized as follows:

1. A formal paper is made out by somebody (family physician?) notifying a public officer that a person is insane and requires asylum care.

2. This official directs two physicians (more than likely political friends of his), in a formal paper to each, to examine the patient.

3. The two physicians make out two documents certifying to the insanity.

4. If the public officer notified be a justice of the peace or superintendent of the poor, he too must visit the patient, to corroborate the diagnosis of the doctors and make out a formal paper as to his findings. He then presents his own and the doctors' papers to a judge of a court of record.

5. Such judge may then cause the patient to be brought into court, or visit him himself also, or try him by a jury. If he does neither of these, he makes out three more papers (one announcing to the sick man that he is a lunatic and about to be sent to an asylum; this must be served upon the invalid personally. The other two are an order committing him to some institution, and a warrant for his transfer to the asylum).

6. Then the asylum officer gives formal notice to the judge of the receipt of the patient, and the judge files this with the county clerk.

7. The judge causes copies of the medical certificates to be filed with the county clerk. He must also take proof as to the estate of the patient, and file a certificate upon the facts with the county clerk.

It would be far better to leave the law of commitment as it now is, and introduce the other features of the Gallup Lunacy Bill into our present system. The tendency should be not to the multiplication of formalities in committing the insane to hospitals, but to the simplification of methods, in order that the insane may be early treated for the disordered condition of their brains. It is to the future we must look for legislation which shall open all of our general hospitals to the insane, and which shall make ingress and egress easy to and from all of our insane institutions. In this way will the greatest good be accomplished, and no harm from wrongful detention ever result.

PERISCOPE.

BY G. M. HAMMOND, M. D., LOUISE FISKE-BRYSON, M. D.,
AND GRACE PECKHAM, M. D.

NOTE ON THE PATHOLOGICAL ANATOMY OF THE SO-CALLED ESSENTIAL EPILEPSY. By M. le Dr. Chastin (Journ. des Conn. Méd., March 21, 1889).

The author had the opportunity of examining the brains of five epileptics. Four of them presented lesions which are described under the title of atrophic sclerosis. Macroscopically, the convolutions were shrunken, small, hard, smooth or slightly roughened, and not adherent to the pia mater. This pathological transformation was not uniform over the cortex of the brain; a considerable area was observed to be healthy, according to the brain examined, but the bulb and the cornua Ammonis were always involved.

The microscopical examination of this sclerosis demonstrated that the fundamental lesion was due to the presence of numberless stiff fibres of an undetermined length, which had invaded the cerebral tissue, particularly the gray substance. In the normal state the first layer of the cortex contains a few spider-shaped cells with few visible prolongations. Here, on the contrary, the first layer is formed by a bundle of fibres, whose direction is nearly parallel to the surface of the brain, and which can be distinctly seen originating by hypertrophied prolongations from numerous cells. The author exhibited one preparation in which this change had invaded all the layers, leaving intact, however, the nerve cells and the vessels. The fibres in certain places form in the substance of the cortex a net-work at the nodular points of which neuroglia cells are found. This net-work forms large, compact bundles, which are unquestionably developed from the fibres. The vessels which existed

presented no traces of inflammation; there was, at a few points, a hyaline exudation into the walls of the capillaries. The examination of the motor regions of the brain, which were about as hard as an olive, demonstrated that the neo-formation had begun in the neuroglia fibres.

The fifth brain presented no appreciable lesion to the naked eye. Under the microscope, however, in the region of the paracentral lobule, the same neuroglia changes were observed that were found in the other brains.

The author reaches the following conclusions:

1. Certain lesions, described under the name of cerebral sclerosis, are due, in many instances, to proliferation of the connective-tissue, particularly of the fibres of the neuroglia. For these cases the name of *neuroglia sclerosis* or *glioma* was proposed.

2. The induration of some portions of the brain, particularly of the cornua Ammonis and the olives, has been known for a long time in connection with epilepsy. This induration is the external evidence of the internal proliferation of the neuroglia. *When there are no macroscopical lesions, the same neuro-pathological fundamental process can nevertheless be recognized.*

G. M. H.

THERAPEUTICS OF THE NERVOUS SYSTEM.

HYSTERICAL CHOREA: CRISES CHARACTERIZED BY HYPOCHONDRIA, FOLLOWED BY AN IMPERIOUS DESIRE TO WALK, TO CRY, AND TO SING; CURED BY COPPER USED EXTERNALLY AND INTERNALLY.

Dr. Moricourt, at the Société Médicale (Journ. des Conn. Méd., April 18, 1889), reports the following case: The patient, a woman fifty years of age, had had these nervous crises for five years, ever since the death of her child. She had been subject for a long time to attacks of migraine, accompanied by nausea, which had diminished in frequency with the advent of the nervous crises. One of her sisters had genuine hysterical attacks. The crises usually came on between two and five o'clock in the morning. They

generally began with melancholy thoughts and with a desire to cry, frequently with headache, anorexia, nausea, and diarrhœa. This was soon followed by trembling in all the limbs, then by an imperious desire to walk, to cry, to sing, and to embrace people. These crises lasted about five hours, and occurred from three times in a month to several times in a week. Anæsthesia and analgesia were present on the outer surface of both forearms. An armllet, composed of four disks of steel, was applied to each forearm. At the end of ten minutes the patient experienced slight warmth in the right arm, which soon passed to the left arm, oscillated from one arm to the other, and then disappeared. The anæsthesia and analgesia were diminished, and the muscular strength also to a slight extent. The next day two armllets of copper were applied in the same manner. The patient experienced sensations of pain and heat much more acutely than with the steel; analgesia was diminished, while anæsthesia was increased. Zinc, tin, gold, silver, and aluminium were successively tried without result.

Dr. Moricourt therefore prescribed *eau d'Orezza* and external applications of copper. Under this treatment the intervals between the crises became longer, the appetite improved, and the menstruation, which had been absent for three months, returned. Shortly afterward the patient, having neglected the treatment for two days, was seized with another crisis, but much less severe than formerly. The water was then discontinued, and, in its place, pilules of sulphate of copper were given. Each pill contained one milligramme of sulphate of copper. The dose was two pills four times a day.

This treatment was continued from the 28th of January till the 23d of March. In this time the sensibility became normal on both sides, menstruation returned, and the crises disappeared.

G. M. H.

TREPHINING FOR CEREBRAL ABSCESS.

M. Terrillon, at a meeting of the Société de Chirurgie, July 3, 1889, reported the case of a child, thirteen years of age, who, following an attack of typhoid fever, presented a

phlegmonous swelling over the left temporal region which was followed by aphasia and by paralysis of the right side of the face and the right arm. An incision into this subperiosteal abscess in the temporal fossa was not followed by any amelioration of the cerebral symptoms. The next day trephining was performed, and a button of bone was removed from over the area of the centre for the upper extremity, and a lesion was sought for which M. Terrillon believed would be found between the bone and the dura; but nothing was discovered.

Suspecting a cerebral abscess, three exploratory punctures were made, instead of an incision, as the operator did not believe that the rules laid down for cranio-cerebral topography in the adult applied equally as well to the child's brain. On the third puncture an abscess was found, which was freely opened. Following the evacuation of the contents of the abscess, the paralysis almost completely disappeared. Three days after the operation, meningo-encephalitis developed, and the patient died.

G. M. H.

OBSERVATIONS AND THEORIES RELATING TO HYPNOTISM.

In the *Gazette Degli Ospitale* are reported several interesting cases in which good results have been obtained by means of hypnotism.

The following case of nervous vomiting is reported by Dr. A. Colombi, in the *Journal* of March 31, 1889. The patient was a girl of twelve, who for a month had been affected with nervous vomiting occurring suddenly after each meal. She was irritable and showed a great lack of strength. She was hypnotized twice by means of fixing her eyes on a clinical thermometer for a few minutes. The vomiting and general weakness disappeared after the second treatment, and the irritability likewise, the child becoming happy and contented.

Dr. G. Ficano, in the same journal, August 11, 1889, publishes a case of hysterical cough cured by what he calls "moral" influence. The patient, a young woman, had suffered with a severe cough for several months. Her par-

oxysms were frequent and appeared like laryngismus stridulous, and the cough sounded like the barking of a large dog. There was no expectoration.

An examination with the laryngoscope was attended with such difficulty that the physician brought in an old patient to demonstrate the process ; the latter, owing to the medication had such a spasm of the glottis as to greatly alarm the young woman, after which it was noticed that her cough decreased in frequency and in intensity. Seeing this the physician threatened her with an operation if the cough did not cease. This, together with a couple of examinations with laryngoscope, and the application of five per cent. solution of cocaine, resulted in a cure. The case is not remarkable or unusual, but its interest for the present time results in the pointing out of the relation which exists between a strong moral influence acting on the physical in the same manner as an hypnotic suggestion. Suggestion, the writer goes on to say, is none other than a very powerful moral agent acting not alone on the intelligence, but also on the body, on the physical.

Quierolo reports in the *Gazzetta Degli Ospitali* of September 4, 1889, a very interesting case of a girl sixteen years old who had a great horror and aversion to food. She would have a spasm of terror every time nourishment was offered to her in any form. No argument or persuasion was of any avail and she was soon reduced to skin and bones. This was the very abnormal symptom, her mental qualities, her affections and desires were perfectly well balanced. She wished ardently to be cured but was dominated by this paralyzing terror of food.

The physician encountered the greatest difficulty in hypnotizing her. Few would have persevered as he did. He tried every method known for a month and a half. He even hypnotized a patient before her. Finally, he made her feel somewhat sleepy by placing a magnet before her eyes. After several sittings hypnosis was obtained, before this every suggestion to eat was received by shaking the head. This time the suggestion was made that the next day at eleven she would come to the physician's house and would

eat, and that food would taste good to her. An attempt was made to make her eat before that time with no success. Toward eleven she was dressed and led to the house, seated at a table, and without resistance began in a mechanical way, to carry her fork to her mouth and continued to eat during the meal.

She was cured in a month and a half from the day of the first suggestion, having passed three years in the miserable condition described.

The author has already cited claims that hypnotism should be more used as a therapeutic agent. Doctor Giacomo Lombroso's article in *Lo Sperimentale* of June, 1889, on this very subject is most just and able. It is entitled *Hypnotism as a Curative Measure*. He first discusses the difference of opinion between the Parisian School (Charcot) and that of Nancy (Bernheim) as to whether hypnotism is to be considered a phenomenon of suggestion, or whether it is produced in its three stages of lethargy, catalepsy, and somnambulism spontaneously. He cites arguments and cases which he thinks amount to a demonstration of the fact that hypnotism is in reality a neurosis which may be provoked by suggestion, or spontaneously, or with some manœuvre without suggestion of any sort. The doctrine adopted at Salpêtrière, by Charcot, is not a classification of the disease but an analysis. The complete forms are rare, but can be produced artificially from the incomplete forms which are common.

The susceptibility to hypnotism is less than that established by Bernheim. He thinks it 50 per cent. of the neuropathic.

Dr. Lombroso considers his subject under two heads.

1st. What are the dangerous effects of hypnotism.

2nd. What curative action has it in different diseases.

His first point is one of great importance and is apt to be lost sight of in the promiscuous experimentations which are constantly taking place. He speaks of the indirect injury which results from the knowledge to the laity that there exists a method by which it is possible to render the will of the individual inert, to constrain him to do and to

think according to the wishes of another, giving aims as has already been the case to a number of complicated medico legal questions.

Still more important are the dangers which are the direct outcome of its use on the individual. They may be said to be immediate and remote, the latter being by far the most frequent. He gives an instance of a young girl who lay for many hours in a lethargic sleep, from which it was impossible to awaken her. Flagellations, electricity, stimulation of every kind were used, until at last the awakening was spontaneous. The extremities were cold, and there was extreme pallor.

In the case of another young girl afflicted with hysterical paralysis, whom he attempted to cure with hypnotism, at the first attempt there were convulsive phenomena, accompanied with vomiting and hiccoughing. She was removed to a hospital. The convulsions, which were very severe, lasted three hours.

The immediate ill effects of hypnotism are not nearly as frequent as the remote. Some who are hypnotized for the first time remain in a dazed condition, and it is several days before they regain their natural state. After repeated hypnotism they change their character: some are melancholic, others are a prey to morbid excitation. Many fall an easy victim to diseases similar to those of which they have been cured—a predisposition to contagion. Others have a mania to simulate diseases carried to a greater degree than in the hysterical. Others assert themselves in a bizarre way, as if seeking compensation for the violence done to their will, or will attempt to gain their wishes in any manner possible. To the psychic state of these post-hypnotic conditions he gives the name of *neurosis of the hypnotized*. He adds that, since hypnotism is a disease which acts directly upon the brain, it is not strange that it leaves some trace behind.

The author does not deny that, as a therapeutic agent, it has had at times the most happy curative effects. Insanity, paralysis, contractures, spasms, neuralgia, have been cured by it. He cites several interesting cases of cures.

He concludes from his discussion of the subject with the

following remark, which, it seems to us, should be borne in mind by whomever attempts to use hypnotism :

“Hypnotism should be used solely in those cases in which the gravity and the pertinacity of the malady indicate it, and only then after the use of every means of cure known to our art.”

G. P.

25 Madison Avenue.

HYSTERICAL CHOREA.

In the *British Medical Journal*, of July 6, 1889, P. Blaikie Smith reports a case of this kind in a woman, aged twenty-two, with the following symptoms when first seen : Eyes closed, face averted, shoulders and arms in constant movement. Each shoulder was alternately either violently raised or brought forward, and then suddenly lowered or retracted, while the arms were constantly rotated inward or energetically flexed. The arm and shoulder movements seemed to bear no relation to each other, though both sets of movements were liable to sudden and unaccountable exacerbations. There were no spasms of muscles about the neck, no contortions of the face, and speech was unaffected. The tongue was protruded quietly, and remained out for inspection until the patient was told to withdraw it. The grasp of both hands, notably the right, was much impaired. Superficial reflexes of the legs and trunk were greatly diminished ; the knee-jerks were much exaggerated, and capable of being produced by tapping the rectus tendon on the front of either thigh. Rectus clonus was present on both sides, ankle clonus absent, the legs free from spasm, heart-sounds normal, and heart-action regular.

It soon became evident that the spasmodic movements were performed with a certain regularity, that they ceased during sleep, were intensified by emotion, yet could in a measure be controlled. The patient could feed herself, could knit, could write, but not legibly, on account of her violent contortions, but could not sew. The body swayed when walking, and progress was erratic. The mind was clear, and the woman generally cheerful, though there were occasional fits of crying.

This condition obtained for about three weeks in varying degree. The reflexes were not constant, sometimes normal, sometimes exaggerated, seldom equal on both sides.

There was nothing important in the family history, though the origin of the trouble was significant. Five years before she had acted as nurse to a child suffering from chorea, and, three months before coming under observation, another child in the patient's neighborhood had the same complaint. She had had a serious fright from a dog, and afterward suffered from nervousness. But the convulsive trouble came on four days before entering the infirmary, when a violent pain seized the right foot, and the "shakings" began. Large doses of bromide of potassium, in combination with tincture of asafœtida and subsequently valerianate of zinc in the form of a pill, produced no improvement. When internal treatment was changed to four minims of liquor arsenicalis, the effect was marvelous. In two days the patient was as quiet as any one when attention was not especially directed toward herself. Emotion would bring on transient convulsive movements. She soon regained her normal power. The varying reflexes, the erratic and inconsistent symptoms, suggested hysterical chorea, together with the history given, which supposition was verified by the sudden cessation of symptoms, that had lasted a month, through the agency of a fresh plan of treatment.

L. F. B.

WAKEFULNESS IN NEURASTHENIA.

A wide range of opinion on the management of this condition found expression at a recent meeting of the Epidemiological Association; and the *New York Medical Journal* thus sums up the evidence:

"The use of drugs, with the exception of sulphonal, perhaps, did not find much favor with the members. Some of them had found that their patients of this class slept when they were at the seaside, while others recommended the Colorado atmosphere. Some patients had been found

to be able to sleep at sea, but not on land. The weight of evidence seemed to favor the resort to mountain air for patients who were anæmic, with a presumption in favor of sea air for those who were plethoric. Dr. Solly, of Colorado Springs, has found that a large proportion of anæmic neurasthenics find sleep on the mountain heights, but this cannot be said of the entire class. It is not improbable that other conditions besides those of climate enter into the account where the patient travels from our Eastern cities to the Rocky Mountains in pursuit of sleep. The jaded matron leaves the worries of the household, and the business man, broken down by the rush of daily cares, finds many things changed besides the atmosphere among the far Western altitudes. Still, as a rule, the climate gets all the praise, when an improvement takes place. Business men from the East report a larger percentage of recoveries than the matrons, however, probably because fewer of their anxieties can follow them. Improvement in the assimilation of food, it should not be forgotten, goes a great way toward sleep-production in those who are affected with derangement of the nervous system; and this is one of the frequent accompaniments of any change of scene and environment. Not that there is always any marked increase of appetite or in the amount of food taken, but there is an appropriation of the food by the nervous centres, to their consequent strengthening. It is often a prominent feature in neurasthenia that the food may be taken in and digested fairly well, but stops short somewhere in its distribution to the tissues and is largely wasted. Ordinarily, when this waste ceases there is a corresponding abatement of wakefulness and other neurotic symptoms."—*Bost. Med. and Surg. Jour.*

THE DILATATION OF THE PUPIL IN LOCOMOTOR-ATAXIA.

Dr. Angel Money (*Lancet*) has observed a dilatation of the pupil in locomotor-ataxia in which they did not contract to light.

According to his observations, this dilatation only occurs when the light employed in the search for th Argyll-

Robertson symptom is intense. He thinks it is due to intense heat and light acting through the conjunctiva on the fifth nerve, similar to the action that takes place when the nape of the neck is irritated. The pupils in these cases of locomotor-ataxia reply in the same way, though not as much so. In the cases in which these phenomena occur, the pupils were not very much contracted.

A modern "Rip Van Winkle" is said to reside, or rather sleep, in Utica, Minnesota.

He has been sleeping most of the time for the past twelve years, and has only awakened occasionally for a day. His latest nap is said to have lasted thus far two years.—*Canada Med Record*.

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, Tuesday Evening, October 1st, 1889.

President DR. G. W. JACOBY in the Chair.

Dr. A. A. BOYER presented a case of

LESION OF THE PONS,

with history, of which the following is a brief synopsis:

C. W. B., age 45, male, syphilitic, in June, 1888, while at work, suddenly felt a sensation at the back of his head as if he had been shot. The shock was not severe enough to cause him to fall or lose consciousness. It was followed immediately by intense headache. Later, nausea, vomiting and insomnia became prominent symptoms for six weeks, and he was treated for gastritis. There was then an interval of two weeks without these symptoms, after which they returned in greater severity. August 14th his left thigh suddenly grew cold and numb; then it extended to his foot, and during the night spread up the trunk, left arm and left face. In the morning there were anæsthesia and paralysis of the whole left side of the body. On examination eight months later he had hemiplegic gait, incoördination of the left arm, good equilibration, paresis of the left leg, paralysis of the left arm, drooping of left side of mouth, some atrophy from disuse on the left side, hemianæsthesia and hemi-analgesia. The lesion would, therefore, seem to be pretty definitely located in the pons, to the right of the median line, above the line of Gubler, and involving the fillet, the reticular formation and pyramidal tract. The writer thought a lesion here would be high enough to produce paralysis of the facial muscles of the opposite side, and low enough down to leave unaffected the

ocular nerves. He believed the most reasonable theory as to the nature of the lesion was thrombosis, as a result of obliterating endarteritis or hemorrhage. It could not be a tumor.

Dr. BIRDSALL saw no necessity for assuming a lesion of the pons to explain the symptoms, which seemed to be wholly unilateral. He usually expected something particularly characteristic in Pontic lesions, such as alternating paralysis. He thought a capsular lesion would account for all the symptoms in this case, and the absence of other pontic features would strengthen that view.

Dr. STARR agreed with Dr. Birdsall as to the localization of the lesion. He thought the case an ordinary hemiplegia with a lesion in the internal capsule. Unless there were certain cranial nervous lesions, alternating paralysis, or some other characteristic symptoms, there was no need to locate the disease in the pons.

Dr. BOYER, in closing the discussion, said he had been led to locate the lesion in the pons, because there was no loss of consciousness at the onset of the attack, although very decided and varied paralysis were immediately developed. In his opinion this pointed strongly to a minute lesion, and nowhere above the pons would a small lesion be likely to produce such wide results. The incoördination now remaining after the disappearance of most of the motor symptoms indicated a lesion of the fillet or of the commissural fibres in its vicinity.

Dr. L. C. GRAY then read a paper, entitled:

THE CURABILITY OF LOCOMOTOR ATAXIA.

He spoke first of the modern knowledge of the pathology, going at some length into the question of the anatomical side and the histological details. He regarded the original focus of disease as being in the nature of a cellular or inflammatory alteration of the cord, sometimes possibly a meningitis, from either of which the ascending secondary degeneration started. Erb and Schultze had reported a case of indubitable locomotor ataxia, examined microscopically, in which the symptoms had disappeared almost entirely for some twelve years. In view of this, it was to

be affirmed that there was not a positive case on record, although it was equally unquestionable that many cases of great improvement had been reported, more especially among those with a history of syphilitic injection.

Dr. Gray then read a number of such cases, but the so-called "cures" were to be found in those in whom the locomotor ataxia was associated with intra-cranial syphilis, or with syphilitic spinal meningitis, or with a syphilitic history, and exceptionally in the non-syphilitic cases. Dr. Gray narrated a case of his own, illustrating the association of locomotor ataxia with general paresis, the mental hebetude of the latter disease causing an apparent improvement in the symptoms of the former. Dr. Gray then spoke of the different means by which the so-called "cures" had been effected—mercury and the iodides, nitrate of silver, nerve stretching, rest, galvanism and suspension.

Dr. DANA said that we have changed our conceptions as to the variations in clinical types and as to the pathological lesions of locomotor ataxia. Probably ninety per cent of our cases are typical and answer to the classic descriptions of the disease. But there is a minority of cases which have a non-typical manifestation; types with optic atrophy, with latent spinal symptoms, with spastic symptoms, with peripheral lesions, etc., and the prognosis is different in these forms. For instance, the prognosis is not bad in the spastic variety, but exceedingly so where there is rapid emaciation of the lower extremities. He had recently examined his notes of cases, and the results of treatment in fifty-six cases (twenty-two syphilitic) had been as follows:

- 10 very much improved (3 syphilitic).
- 6 improved (4 syphilitic).
- 14 stationary (3 syphilitic).
- 13 progressive (8 syphilitic); 2 fatal.
- 10 doubtful or unknown.

He believed locomotive ataxia to be a degenerative disorder, affecting nerve fibres primarily, not beginning at a single focus, but at several different places at various times.

In order to cure the disease we should have to discover a drug which would stop this degenerative process. As to suspension, he had tried it in 22 cases, with 600 suspensions, since last April. Of these, 4 were very much improved, 6 improved, 6 unimproved, and 6 discontinued the treatment. He thought there was no question as to very marked benefit being produced by suspension.

Dr. BIRDSALL, upon reading the title of Dr. Gray's paper, had been led to think that some new view of the hopefulness of cure was to be introduced by the author, but he coincided with the writer in the opinion he had expressed as to the incurability of the disease. Various physicians entertained different ideas as to what should be termed a *cure* of a disease. Some considered patients practically cured where the pathological process had been arrested under treatment, although many symptoms might remain. If this were true, possibly some cases of locomoto ataxia might be called to a certain extent cured. In one of his cases ataxia had disappeared and had not now returned in nine years, although he had of late suffered from various crisis, gastric and laryngeal, and some cerebral symptoms such as loss of consciousness. For five years the active symptoms had been referred to the trunk and upper extremities. It was a syphilitic case. He had another case where the tendon reflex had returned after abolition for many months, and ataxia had also disappeared; but pains and numbness subsequently became manifest in the upper extremities. He would not himself speak of any case as cured, but only as arrested in its progress. It was well that we are able to recognize the disease much earlier than formerly, and have better opportunities for early treatment. Our measures should be chiefly hygienic, the regulations of diet, avoiding of excesses, diminishing of labor, etc. He had tried suspension in a few cases but with no favorable results. At the same time, he did not deem it wise to throw it as yet altogether aside. He had seen injurious effects from large doses of potassic iodide, and in his opinion tabetic cases were not so well able to tolerate this drug as some suffering from other disorders. We must

modify our opinion somewhat of the varieties of tabes, since Déjerine and others had discovered peripheral nerves lesions in some cases of locomotor ataxia.

Dr. STARR said, with regard to the difficulty of differential diagnosis between peripheral and cerebral cases, we must judge from the order of occurrence of symptoms as well as from the symptoms themselves. He referred to the paper of Déjerine, where it was shown that peripheral cases are distinguishable by the rapidity of their development as contrasted with those of cerebral origin. Alcoholic, lead and arsenical disorders could scarcely be mistaken for actual locomotor ataxia, for a sharp line can be drawn between the two; and even in the cases whose cause is obscure, the course is much more rapid in neuritis.

Some seven years ago Dr. Austin Flint read a paper in this Academy upon the self-limitation of phthisis. A similar description might apply to locomotor ataxia also. It is possible that the sclerosis of the cord may be a protective process; an effort of nature to arrest or provide against the effects of disease. A pathologist in this city has taught for two years that connective tissue is thrown out by nature as a defence. Thus, locomotor ataxia may also be in a self-limited disease like phthisis in many instances, although in the majority of cases it is not.

His own records showed some 25 cases seen in the last four years, 17 of which are carefully detailed. In 9 of the 17 there had been periods of non-progression at times, while in 8 there had been steady advance in spite of treatment; but as similar treatment had been employed in all of these cases, the temporary arrest of the disease in some could not be ascribed to the therapeutics. He agreed with Dr. Dana that eye cases manifest atypical symptoms. In three of his cases with blindness there had been no progress subsequent to the failure of sight. He thought it worth while to try the specific treatment in patients with a history of syphilis, but he had little faith in its efficacy, as in his experience, as in that of continental observers, it afforded less benefit in locomotor ataxia than in other specific nervous affections. He employed usually small doses of arsenic

and biniodide of mercury, which is the English treatment.

In 13 cases treated by suspension at the Vanderbilt Clinic, tabulated by Dr. Peterson, there were 7 cases of locomotor ataxia. There had been distinct improvement in 2 cases only, and none at all in 4, while in 2 the results had been bad; the suspension producing syncope, nausea and vomiting, severe pain and enuresis at different times. It might be stated, therefore, that a small proportion only of cases had been improved by suspension.

Dr. SACHS had observed in two cases the disappearance of the cardinal symptoms of locomotor ataxia without treatment. The first was as follows:

B. L., merchant, age 48; seen August 18th, 1886. Complained of retching with dizziness, a numbness of left arm and unsteadiness in walking. Both knee-jerks were absent and could not be elicited with Jendrassik's method. There was slight swaying with eyes closed, and the patient complained of a feeling as though his drawers were too tight around the waist. He had made the diagnosis of tabes incipiens, but with some hesitation, however, for the general condition of the patient was very good. After two months a slight knee-jerk had returned on the right side, and a few months later the left was recovered. Three years had now elapsed since the observation of these symptoms, and the man was in perfect health at the present day. He believed it to have been a functional depression of the cord due to over-work. A second case had all the symptoms of tabes, except that the knee-jerk was absent on one side only. He had the Argyll-Robertson pupil and the Romberg and girdle symptoms. He had seen him four months ago and all of the symptoms had diminished.

In syphilis a simple specific spinal meningitis might simulate a posterior sclerosis, and this should be borne in mind when cases improve under treatment.

He mentioned another case diagnosticated as locomotor ataxia by Erb twelve years ago, now living in Utah, which has been stationary for five years. As far as actual curability is concerned, he could not speak of that unless a progressive form had been arrested. He would classify cases

into optic and spinal types. As to suspension, he had found it unsatisfactory, and had been unfavorably impressed with it. It had seemed to him better in spastic cases, such as myelitis.

Dr. WAITZFELDER had been using suspension in a case of spastic paralegia, and it had made that case worse, as well as several other cases of myelitis; but he had noted considerable improvement in gait in several patients with locomotor ataxia who had been subjected to this method of treatment.

Dr. LESZYNSKY had observed a few favorable results in the employment of suspension in spinal cases. He had also used it in a case of paralysis-agitans three or four times with good effect.

Dr. GRAY then closed the discussion. He said that Fournier's and Rumpf's specific cases showed great improvement under treatment, but this had not been his own experience. He believed there were certain cases where there would be great difficulty in distinguishing peripheral from central symptoms. As to the matter of self-limitations mentioned by Dr. Starr, he saw no analogy between tabes and phthisis. In treatment he preferred to follow French authors and employ inunction in specific cases rather than potassic iodide, for the results were much better.

FREDERICK PETERSON, M. D.,

Secretary.

AMERICAN NEUROLOGICAL ASSOCIATION TRANSACTIONS.

DR. B. SACHS, of New York, read a paper entitled
POLIOENCEPHALITIS SUPERIOR (NUCLEAR OPHTHALMO-
PLEGIA) AND POLIOMYELITIS.

It is a rare experience in neurological matters to have the pathology of a disease unravelled as quickly as was done in the case of those clinical groups of symptoms which we know as ophthalmoplegia externa and interna.

The paralysis of the muscular apparatus of the eye was soon discovered to be due, in most cases, to a lesion or lesions affecting the nuclei of the nerves which govern the various ocular muscles. In this nuclear paralysis, the nuclei of the oculo-motor nerves play a most important *role*, though the nuclei of the fourth and sixth nerves are involved frequently enough.

The relation of the bulbar process to poliomyelitis was firmly proved by cases of bulbar paralysis which were associated with symptoms resembling those of a progressive muscular atrophy or a chronic anterior poliomyelitis, and furthermore by cases of typical progressive muscular atrophy which, in their terminal stages, developed bulbar symptoms.

On the strength of this clinical analogy, Hutchinson, Mauther, and Birdsall were struck with the pathological resemblances between the diseases affecting the ocular and spinal nuclei; and Wernicke proposed to call the affection of the oculo-motor nuclei a *polioencephalitis superior*, whence it followed that the bulbar paralysis might well be styled *polioencephalitis inferior*. While the analogy with poliomyelitis had been proven beyond the shadow of doubt for the bulbar cases, Wernicke's theory needed further proof as regards the cases of total ophthalmoplegia externa and interna. Heretofore but one case has been recorded (by Seeligmüller) in which the symptoms of a chronic poliomyelitis were associated with those of *polioencephalitis superior*, and with the exception of the cases of Henoch and Buzzard, in which an ocular (nuclear) paralysis occurred in the course of an acute poliomyelitis anterior, I know of no cases which exhibit this interesting association of symptoms.

The history of this case is as follows :

H. M., aged forty, is a man of robust build, unusually intelligent, and one who has experienced all the vicissitudes of life. He was born in this city, and has been married fourteen years. Has one brother living and healthy, and one brother who died from want of water, as the patient says, on the desert of Arizona. Both parents are dead; the

father died of yellow fever in New Orleans in 1858 ; the mother died of a paralysis which lasted seven or eight years and began by turning-in of both feet. The paralysis of the legs became complete ; she died at the age of seventy-four. The patient went to school at Syracuse, N. Y. As a boy he had frequent "bilious" attacks associated with headaches and vomiting, was otherwise in robust health. He attended school until the age of twelve ; went to New Mexico at the age of fourteen, and there learned a trade.

When sixteen years of age, one day after reading several hours got up to stretch himself, but fell back unconscious against a hot stove, burning the left temple severely—the scar is visible at the present day. He was insensible for several hours and was then put to bed ; knew nothing of what had happened until he saw doctors around ; no paralysis followed. A second similar attack, again after reading, occurred three months later, from which he quickly recovered. A third attack occurred, but the date of this he cannot recall. No further sickness until the age of twenty, when he was in Peru and was steward on a United States steamer. While on shore he fell from a horse, striking the right elbow and injuring the arm. Recovered completely from this fall.

At the age of twenty-five was in Europe ; felt one day a severe pain in the left eye ; engaged passage at once for New York ; reaching there a few weeks later. By that time the left eye was closed. Went to sea again (to Australia on a sailing vessel), and during this trip noticed that the right lid was also beginning to droop. He was treated in Melbourne by electricity, but the condition remained stationary for several months. After that a slight improvement is said to have set in the left eye, but the right eye grew worse ; both pupils were dilated (physician's statement). Had double vision all the time, and small ulcers formed on the left eye. The Australian physicians suspected tumor of the brain. He had severe headaches at the time ; the left eye became inflamed and was in such a bad condition that the physician advised enucleation, but the patient objected. After a short trip at sea the inflammatory condition was at an end.

It was about this time that he began to be suspicious of his legs; for one day, while walking on deck, his right knee gave way. A few days later the same accident occurred. Nevertheless, he joined a ship from Australia to California. When thirty days out he had to refuse duty as steward, as he could not move his right thigh, leg or toe a single inch. The captain ordered hot steam bath and gave him blue pill and black draught. He went to Oregon next—now thirteen years ago. He remembers that when there he could not hold water nor contain feces. His left leg was not affected at any time. Iodide treatment was proposed. Patient objected, on the ground that he had never had any syphilitic affection. In consequence of the paralysis of the right leg he was compelled to go about on crutches. The doctor who examined him found a sensitive point between the shoulder-blades, and ordered blisters, and strychnine internally. In six months' time he was able to walk with the assistance of a stick. He could use the hip and knee-joint, but could not move ankle or toes. The eyes remained in about the same condition; he was not worried by them. He undertook contract work on a railroad in Panama, and there contracted severe malaria.

One year and a half ago he was stricken down with prolonged fever. As soon as he recovered from this he traveled about considerably; finally went to Jacksonville, where he got a thorough drenching, which was followed by severe chill. One day he tried to read the papers but could not see anything. Last year, on his return to New York, he was examined by Dr. E. Grüning, who performed an iredeotomy of the right eye, which did not improve vision. Later on Dr. Grüning raised the left eyelid by operation and restored vision to that eye. Went to Panama in May, 1888, and returned to New York about seven weeks ago.

For the past four weeks he has been an inmate of the Montefiore Home for Chronic Invalids, where I have had the opportunity of studying the case. He denies ever having had gonorrhœa or syphilis, and an examination of his body reveals no symptoms of the latter. He has been

moderate in sexual matters and has never been a hard drinker. Has smoked innumerable cigarettes for years. No loss of consciousness has occurred since the attacks recorded above. Does not suffer from headaches, and but for the condition of his eyes and of his right leg would feel entirely well.

Present Condition.—Strong, well-built man; heart sounds normal; no enlargement of liver; slight enlargement of spleen; other thoracic, and abdominal organs normal. The most striking feature of the patient's appearance is the double ptosis, at present more marked on the right than on the left, in consequence of the operation on the left eyelid. Slight lateral nystagmus of right eye. In this eye, also, maculæ cornæ, old iritis with exclusion of pupil dilated. A transparent, thin membrane has grown upward, covering nearly one-half of left pupil. The results of my examination of the eyes, which were kindly corroborated by Dr. E. Fridenberg, are as follows:

O. D.—Paretic—rectus internus, rectus externus, and inferior oblique. Paralyzed—levator palpebrarum, rectus superior, obliquus superior, and rectus inferior. Associated movements with the left eye do not differ from those attempted singly.

O. S.—Paretic—rectus externus, rectus internus, and superior oblique. Paralyzed—levator palpebrarum, rectus superior, rectus inferior, and obliquus inferior. Paralysis of iris (light reflex abolished), ciliary muscle normal. Accommodation reflex good. Media apparently clear in both eyes. Vision, left eye, $\frac{20}{c}$. Right eye, = 0.

Ophthalmoscopic Examination.—Left papilla normal; right papilla cannot be examined.

No change in the facial distribution. Hearing normal on right side; on left side somewhat diminished, but normal bone conduction. Tongue protruded straight, slight fibrillary movements. Sensation of face and tongue normal in every particular. Smell and taste normal on both sides. The left arm appears to be slightly larger than the right, but grasp is equally strong on both sides. Sensation normal to touch and pain. Distinguishes numbers written on

arm with great ease. No reflexes to be obtained in upper extremities. No difficulty in respiratory or abdominal muscles.

Lower Extremities.—Marked atrophy of right leg from hip downward. Largest circumference on right side; hip, $14\frac{1}{4}$ inches; left side, $19\frac{1}{2}$ inches; right calf, 10 inches; left calf, $12\frac{1}{2}$ inches. Patient can flex knee very little, but cannot move toes of the right foot. Walks by exclusive use of posterior thigh and leg muscles. Muscular excitability lost in right thigh. No disturbance in sensation except apparent diminution of pain sense on inner aspect of right thigh. The left thigh and leg muscles show normal myotatic excitability and absolutely normal sensation. No change to be noted in any respect in leg of left side. There is no ataxia of either leg and none in the upper extremities. No Romberg symptoms. The knee-jerks are lost on both sides and cannot be elicited by Jendrassik's method. All cutaneous reflexes sluggish but present.

The electrical examination reveals no change in any of the muscles of the face, of the upper extremities, or of the trunk, nor in the left leg; but marked degeneration reaction exists in the anterior thigh and leg muscles. The vasti and anterior tibial muscles are atrophied to such an extreme degree that no action could be obtained with currents at command.

This history can be summarized in a few words: A man in perfect health, without any specific alcoholic or hereditary taint, is affected with a slowly-developing paresis or paralysis of all of the ocular muscles. This condition is scarcely fully established before a weakness of the right leg is noticed by giving way of the knee. This weakness is developed into a most marked paralysis, associated with extreme atrophy. The symptoms remain restricted to the right leg, become retrogressive, and have not to this day effected the opposite leg. The arms remain entirely normal. The transitory bladder and rectal symptoms were probably due to an extension of the inflammation of the gray matter, and do not imply, to my mind, the existence

of a transverse myelitis, acute, subacute, or chronic. No other interpretation can be put upon these symptoms, except to say that in the course of a chronic nuclear paralysis of the eye a subacute poliomyelitis set in. Both in the eyes and in the leg the disease developed in the same fashion, and has practically remained stationary for years.

It will hardly be necessary in this paper to prove the diagnosis of subacute poliomyelitis in this case, and, considering the rarity of poliomyelitis in the adult, it would be strange, indeed, if the occurrence of such an affection in the course of a polioencephalitis superior were a mere coincidence. It seems to me to prove positively that the ganglion cells of the anterior horns of the spinal cord are subject to the same pathological changes as the large nuclear cells on the floor of the third and fourth ventricles.

The involvement of the iris in the one eye (the condition of the other could not be examined) takes his case out of the category of cases of ophthalmoplegia externa. According to most authors, an ophthalmoplegia externa, with paralysis of the iris, would compel one to refer the lesion to the base of the brain; but since Westphal and Spitzka have plausibly shown that the nuclei for the accommodation and light reflex lie anteriorly and away from the remaining oculo-motor nuclei, it is readily seen that these nuclei, also, one or both, may be effected by the extension of the inflammatory process. It is in this way that I explain the affection of the iris in this case. Since the accommodation reflex remained normal, it is natural to infer that the ciliary and iris nuclei must be some appreciable distance apart. We must be careful, however, not to be too positive in such assertions, for Thomsen has recorded cases in which there were distinct paralysis of various ocular muscles with only the slightest involvement of a few of the nuclear cells, and, strangest of all, one case of paralysis of associated vision upward, due to a gummatous infiltration of the *oculo-motor root fibres*, whereas the nuclei were found entirely normal. It is for this reason, also, that I believe that the determination of the exact location of the various subdivisions of the oculo-motor nucleus, on clinical grounds

only, has been carried too far. This question can be settled in no other way but by the experimental method, or by noting to what extent clinical and post-mortem records tally.

One other point in the case demands explanation: The knee-jerk is absent on both sides. The first suspicion was that of an accompanying *tabes dorsalis*, as in Westphal's well-known case; but this supposition must be abandoned, since a close examination with this end in view has shown the absence of every other important symptom of *tabes*. The absent reflex on the left side must, therefore, be regarded as the only evidence of the extension of the process in the spinal cord to the left half of the cord; but, at the same time, the normal condition of the muscles, the normal electrical reactions, and the total absence of atrophy prove that that side can be effected but very little.

The chief value of my case is, that it proves the close relationship between the gray matter at the floor of the third and fourth ventricles and the anterior gray horns of the spinal cord.

Wernicke chose the term *polioencephalitis superior* wisely enough; but Strümpell's *polioencephalitis*, a supposed cortical disease, has caused some confusion. Strümpell's theory and disease lack proof, and for the present we need not decide whether we shall have to add a *polioencephalitis suprema* to *polioencephalitis superior*.

Dr. SPITZKA said the report of the case had been so complete that, as there was but one other such case on record, it did not admit of either criticism or comparison. He referred to Thomsen's case of unilateral nuclear paralysis, where there had been gummatous infiltration on both sides, the explanation of which was to him quite impossible.

Dr. SACHS asked if fibres could be traced up to the ciliary nuclei.

Dr. SPITZKA answered in the affirmative, and made black-board drawings illustrative of their course.

HYPEROSTOSIS CRANII.—Dr. W. N. BULLARD, of Boston, then presented for Dr. J. J. PUTNAM a skull which was a remarkable example of this condition. The case had been

reported to the association two years before. The patient was a woman, thirty-one years old at death. The chief symptoms had been headache, broadening of the head, dropping out of the teeth, loss of hearing, and vertigo, beginning gradually some years ago. There had been extreme exophthalmia. There had been no retinal changes. Extensive pachymeningitis had been discovered at the autopsy. There were thinning and atrophy in parts of the skull. The orbital cavities were greatly diminished in size. Virchow considered hyperostosis cranii due to inflammatory changes. In this case probably the inflammation had originated at the ear. Dr. Putnam had a patient now with similar symptoms, in whom the exostosis were first noticed in boyhood, and desired the opinion of members as to the justifiability of removal of certain exostoses for the relief of pain. Dr. Bullard himself thought it might be difficult to determine which of the exostoses produced the pain. Some of the exostoses were very diffuse, and the operation might have to be extensive.

The PRESIDENT believed that the pain would be more apt to originate from basal regions, possibly dural inflammation about the issuing nerves. The jaw in this case was interesting because of its senile conformation and angle, despite the patient's youth.

SPONTANEOUS DEGENERATIVE NEURITIS OF THE BRACHIAL PLEXUS.—Dr. W. M. LESZYNSKY, of New York, read a paper with this title. The patient was a laborer, aged thirty-eight. He had first had pain in the left shoulder, shooting down the arm, which had been ascribed to exposure to wet. There had been then no involvement of the shoulder-joint and the motility of the arm had been unimpaired. All the muscles had reacted well to faradism, except the deltoid, which had been atrophied. There had been no sensory disturbance, but there had been pain on pressure. Gradually other muscles had become paralyzed, until a large number of the arm-muscles had been useless. The paralysis had been accompanied by pain, so excruciating that the patient could not sleep at night. A feeling of numbness had extended from the shoulder down the arm

over the radial distribution, and a gradual anæsthesia analgesia had supervened throughout the same area. The faradaic excitability had disappeared, and there had been galvanic superexcitability. Then he had begun gradually to improve, and would ultimately recover. The case had been remarkable in its severity, in its idiopathic origin, and in the escape of the median and ulnar nerves from the inflammatory process. Not more than one case, had to his knowledge been found in literature.

Dr. PRINCE thought it would be difficult in the early stages of such a case to distinguish it from progressive muscular atrophy of the shoulder type. He recalled a case of his own in which there had been every reason for considering it to be neuritis. It had begun with cramps, such as were observed in writer's cramp, and it had been several years before other symptoms proved the case to be one of progressive muscular atrophy. The most common cause in such cases as the author's was traumatic arthritis, but generally the results were slight.

Dr. W. R. BIRDSALL, of New York, had seen this case before, and was impressed with the idea that it might be a periarthritic affection, but the author's careful study of the case seemed to exclude this. He recollected that at that time there had been some ankylosis of the shoulder-joint in the case.

THE PRESIDENT considered the study of the differing resistances in the healthy and diseased arm made by the author interesting, and asked if any member had had experience in such measurements.

Dr. GRAY had noticed much variation in resistance in many of his patients from day to day.

Dr. M. A. STARR, of New York, had measured the resistance in Basedow's disease, and had found it to vary within a thousand ohms in the same cases from time to time. Electricity was diffused through muscular better than through any other tissue. The chief resistance was in the skin. Probably but little of the current permeated nerves, and hence alterations of nervous tissue would not have much to do with the variations mentioned by the author.

Dr. G. M. HAMMOND, of New York, stated that it was well known that the resistance differed from day to day in animals. He asked if in this case it had been measured for a number of times, and was answered that eight trials had been made.

Dr. BIRDSALL thought the question of resistance had little to do with our study of disease. It was easy to explain the numerous variations by the wide differences in vascularity and moisture of the tissues at different times. It might be due in this case to paralysis affecting the physical condition of the tissues. The saturation of the epidermis by perspiration would explain the variations mentioned by Dr. Gray. He had made measurements in cases of Basedow's disease some time ago, and believed that the diminished resistance found was due to the moisture of the skin. It could not depend on dynamic conditions in the nervous system, but was purely a matter of physics about which there was no great mystery.

Dr. GRAY said the explanation by moisture of the skin was not applicable in his cases. The differences which he had observed had not been owing to the humidity of the atmosphere, although atmospheric conditions might possibly induce dynamic changes in the body.

Dr. SACHS pointed out that on one day the author's patient showed 580 ohms increase on the diseased side, and on another day 1,170 ohms increase on the sound side. Such variations evidently had little to do with the pathological process in the patient. Eulenburg had measured the resistances in cases of paralysis agitans and of Basedow's disease, but without any practical results. As such measurements were very complex, they required exceedingly great care.

Dr. PRINCE objected to the use of the palm of the hand for precise experimentation, owing to the great variability of the thickness of the skin, and consequently of the resistance. He thought the forearm ought to be used.

Dr. LESZYNSKY had not brought forward the question of resistance in this case as a diagnostic symptom. It was increased in the affected arm at every examination. With

the subsidence of the symptoms the resistance gradually diminished, but there was a difference between the sound and morbid sides throughout the disease. As to the question of moisture, there was excessive perspiration upon the paralyzed arm, while the normal arm was dry, in spite of which the resistance was greater upon the former. He thought it better to place the electrode upon the wrist than in the hand. There was no antecedent traumatism in the case; the roughening of the shoulder-joint was the result of the paralysis.

Dr. M. ALLEN STARR, of New York, then presented a specimen of an

INTERPEDUNCULAR MYXOSARCOMA.

It lay in the middle cranial fossa in the median line between the crura cerebri, which it separated. It extended into the lateral ventricles, separating widely the caudate nuclei and optic thalami. The patient was a male, aged twenty-one months at death. At the age of thirteen months, in October last, a lateral nystagmus had been observed in both eyes, varying from time to time. Drs. Knapp and Roosa found a slight pallor of the optic disks, which they considered normal. Later, exophthalmos developed, gradually increasing until death. Convergence of the eyes was impossible, although no paralysis of a cranial nerve was discoverable. The reflexes of the iris were lost. Toward the middle of April, this year, the child became unable to walk. The knee-jerks were exaggerated, there was ankle clonus, and typical spastic rigidity. Finally, the back could not be held up, and later the head could not be supported. There was gradual emaciation. There was no apparent headache. From time to time the scalp was congested. About this time the pallor of the optic disks indicated atrophy. There was no blindness, no hemiopia, no aphasia, as far as could be ascertained in so young a child. Toward the middle of May ataxia of the arms developed, but without loss of muscular power. June 8th, vomiting and Cheyne-Stokes'

respiration came on, and the child died in nine hours. The diagnosis of tumor had been made, but the question of localization was of great interest. Nystagmus was not a localizable symptom, but had been found most frequently in lesions of the corpora quadrigemina. The exophthalmos indicated intracranial pressure; the gait disturbance led one to think of a cerebellar lesion; while the ataxia of the hands pointed to a basilar trouble affecting symmetrically either the pons or the medulla. The question of operative interference had been considered, but he had opposed that proposition because of the difficulty of localizing the tumor. The autopsy proved that the pons was not even pressed upon, and that the cerebellum was normal. There were twenty ounces of fluid in the ventricles. He would ask if there was any localizing value in nystagmus or exophthalmos, and what was the probable cause of the ataxia.

THE PRESIDENT, in referring to the question of ataxia, recalled a case of remarkable bilateral ataxia, with optic atrophy, where a large interpeduncular tumor was found between the crura. He thought such ataxia was to be accounted for by pressure upon the motor tracts or motor nuclei. To him the most puzzling feature of Dr. Starr's case was the absence of blindness. As to the nystagmus, it had as yet, in his opinion, no localizing value. He had seen two cases with lesions of the quadrigeminal bodies, but without nystagmus.

Dr. LESZYNSKY said the child may have had only central vision, which might explain the presence of nystagmus; but Dr. Starr answered that the visional fields were normal.

Dr. SACHS was reminded of Meynert's case of tumor in both optic thalami, with ataxia similar to that of Dr. Starr's case. He thought the thalami might have been pressed upon in the latter, but still was not sure that that would cause the ataxia. He considered nystagmus very frequent in many central disorders of children.

Dr. H. M. LYMAN referred to a case he had seen recently of defective cerebral development in a child where there was also nystagmus.

Dr. GRAY asked how a diagnosis of intracranial tumor had been made so early in this case, and was answered that the diagnosis had not been made until all the symptoms described had made their appearance.

Dr. HAMMOND thought ataxia depended upon injury to the optic thalami or corpora striata, and referred to a case of Weir Michell's in which there was a remarkable unilateral ataxia, with a lesion of the optic thalamus and corpus striatum upon the opposite side.

Dr. LESZYNSKY saw a child, several years ago, with well-marked nystagmus, which disappeared in the course of time. There was no discoverable cause.

The following gentlemen were then elected to active membership : Dr. C. Eugene Riggs, of St. Paul, Minn. ; Dr. H. S. Upson, of Cleveland, O.

Dr. B. SACHS, of New York, presented a paper on the
PERONEAL FORM OF PROGRESSIVE MUSCULAR ATROPHY.

The author reiterated his statement of last year that this form was closely related to Duchenne's type. He gave very full details of the cases of two brothers that had recently come under his observation through the kindness of Dr. Gibney. The boys were thirteen and ten years of age. There was a gradual development of double clubfoot in both at the age of five years, followed by an atrophy proceeding upward, beginning in the leg and toe muscles and spreading to those of the thigh ; in one case also involving muscles of the upper extremities. The knee-jerks were present. In one there was general anæsthesia, in the other paræsthesia. There was full degenerative reaction in some of the muscles in one boy, partial in the other. The progressive wasting rendered treatment of this form of club-foot less satisfactory than that of congenital cases. He would suspect this peroneal form in all cases where acquired club-foot was associated with progressive wasting of the leg muscles, and par-

ticularly if heredity or family occurrence of the disease could also be established.

Dr. SINKLER could recall several similar cases ; one, in particular, of two brothers. But doubtless more would be seen if careful attention were paid to the matter.

Dr. BIRDSALL did not think the presence of knee-jerk so important a diagnostic point as the author seemed to regard it. He had seen a few cases of old poliomyelitis where the paralysis and atrophy were below the knee, and yet the knee-jerks were quite active on both sides. It could not therefore be an essential point in diagnosis.

Dr. BULLARD had also observed the presence of the knee-jerk in old cases of poliomyelitis.

Dr. PRINCE asked if the author had said that the absence of pseudo-hypertrophy was a diagnostic point between this form and primary myopathies, and was answered in the affirmative. He did not consider this true.

Dr. SPITZKA asked if the symmetry and the coincidence of time and intensity as shown in the photographs were always the case, and was answered in the affirmative.

Dr. SINKLER corroborated two of the speakers as to the presence of the knee-jerk in cases of old poliomyelitis, and cited an instance from his own experience.

Dr. GRAY objected to the division of progressive muscular atrophy into groups. Why should there be an arm type, a face type, a leg type ? Such division might be carried out indefinitely. A more useful classification would be upon the pathology of the disease—a division into central, muscular, and peripheral nerve-lesions.

Dr. SACHS said he did not lay great stress upon the presence or absence of the knee-jerk. Yet in extreme atrophy of the vasti from poliomyelitis the knee-jerk was always absent ; and if it were present, he should consider it a case of progressive muscular atrophy. He had himself tried to discard subdivision as much as possible, but the present classification was a clinical necessity. A better might be made when the pathology is more accurately determined. At present there was spinal and non-spinal cases, but there was no certainty as regarded peripheral nerve cases.

Dr. MORTON PRINCE, of Boston, then exhibited some microscopic specimens from the muscles of a case of

PSEUDO-HYPERTROPHY.

The patient was now twenty-eight years of age. The specimens showed a large quantity of connective tissue, hypertrophy of a few fibres, and great atrophy of many of the fibres. There was also great loss of striation, but no fatty or granular degenerations, and no vacuolization.

Reviews.

REFERENCE HANDBOOK OF THE MEDICAL SCIENCES. Vol. VIII. New York: Wm. Wood & Co.

This concluding volume of a valuable work comes as carefully prepared and as elaborately illustrated as any of the others. The volume is mostly an appendix and index. In the appendix is embodied much of interest to the neurological as well as to the general student.

We would draw especial attention to the article on the Gross Anatomy of the Brain, by Prof. Burt G. Wilder, of Cornell University. It is certainly a very complete article and of value to the neurological student. The author's original nomenclature, however, interferes with the utility of the article as a ready reference, and we would consider it somewhat out of place in a "reference handbook" until the time when the terminology used is more universally understood and adopted.

In this same appendix, in an aphoristic article on Terminology, added by Simon H. Gage, Dr. Wilder has given a careful digest of the subject, and in §45 "begs a careful consideration of his plans for terminological improvement, because they are based upon unbiased study of nearly all previous publications; . . . because the actual disturbance of existing order of things is kept at a minimum; and because the practical availability of the terms has been demonstrated with hundreds of students in both university and medical school."

Prof. Wilder deserves the support of the profession, undoubtedly, for his wonderful work in a much-needed field. That the work of the student of anatomy will be much aided if the existing terminology were simplified, is without a doubt; and much more so, as Dr. Wilder says finally in a list of reasons, why terms were best if they "remained constant, rather than varied."

We would commend highly also the companion articles—Methods of preparing the Brain, and Brain Malformations—which are morphologically instructive.

William Browning has contributed a good and well-illustrated article on the Vessels of the Brain.

Leopold Putzel presents an admirable monograph on Acute and Chronic Myelitis.

Frederick Peterson, an article on Cephalocele.

There are two valuable, concise, yet fully descriptive articles, by Dr. E. C. Spitzka, on the Histology of the Brain, and Anatomy of the Spinal Cord.

The editor, Dr. Albert H. Buck, has finished a creditable work; especially is this observed in his selection of the brightest and best-known writers from the neurological fields of medicine at home, without foreign aid. The result is that the neurological part of this work is up to date, readable, and crowded with information of practical value.—C. H. B.

**Which is the Most Powerful ?
and the Most Reliable ?
of All Pepsins ■**

Merck's Pepsin 1:2000

SCALE OR POWDER

SEE "MERCK'S INDEX," PAGES 106 AND 167

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

THE SOMEWHAT FREQUENT OCCURRENCE OF
DEGENERATIVE DISEASES OF THE NERVOUS
SYSTEM (TABES DORSALIS AND DISSEMI-
NATED SCLEROSIS) IN PERSONS SUFFERING
FROM MALARIA.¹

By MORTON PRINCE,

Physician for Nervous Diseases, Boston City Hospital.

FOR some time past my attention has been attracted to the number of patients suffering from degenerative diseases of the central nervous system, preferably tabes dorsalis and disseminated sclerosis, who had at an earlier period contracted intermittent fever *and were still subject to malarial attacks*. More lately I have noted all cases coming under my observation where the two diseases coexisted, and have now notes of a number of cases which I will refer to in this paper.

For myself, though I do not think this evidence can be taken as decisive of an ætiological law, or at least as showing any great frequency of a connection of this sort, yet I give it with the hope of eliciting the personal experience of others and of drawing attention to the subject for purposes of future observation.

We in Massachusetts do not have many opportunities of seeing malarial fever, and therefore I have hoped that those

¹ Read at the annual meeting of the American Neurological Association, June, 1889.

of you who come from malarial districts may be able to confirm or otherwise explain my own limited observations.

I am further led to introduce the subject here because there are certain peculiar and practical considerations connected with it which render it one of great importance and make it desirable that the question should be settled if possible. To them I shall refer later.

It is not necessary before a society of this kind to describe the cases *in extenso*, a brief reference to the salient features of each will be sufficient, dividing the cases into natural groups.

GROUP I.—TABES DORSALIS.

Case I., M. H. R.—Typical case of locomotor ataxia, knee-jerks absent—static and locomotor ataxy—no appreciable objective anæsthesia, but decided loss of muscular sense in legs, Argyll-Robertson's pupils, paresis of bladder, lightning pains, subjective numbness of hands and feet, etc.

History.—First malarial attack in 1862. Has had similar attacks ever since and still continues to have them. He never goes a month without an attack, and sometimes they recur at intervals of a week or fortnight. There is one very suggestive thing about these malarial attacks, and that is the way in which they begin. The first symptom, as the patient describes them, is shooting-pains down his legs and arms into his fingers, accompanied by a terrible dull feeling in his head. These pains "are very severe, just like a knife or like lightning going through a cloud." Then in the course of half a day or a day the rigor comes on. "Sometimes," to continue his phraseology, "you would think he would shake the bed to pieces." Then fever comes on; he feels as if he would burn up. This in turn is succeeded by cold sweating, and he is left so weak he can hardly stand. In short, he has typical attacks of malaria, always preceded by tabetic crises. He is generally disabled for a week or two. On the following day, or third day or so, he is liable to a second attack and often a third. After the chill comes on the lightning pains are ameliorated,

but his legs then feel numb and dead. For awhile his legs are so clumsy he can't go up and down stairs; then he improves till the next attack.

As to the time of development of the spinal symptoms, the lightning pains first came on shortly after the first attack of chills and fever—say six to eight months. They have increased in severity ever since. The clumsiness of legs was first noticed twelve to fifteen years ago in the following way: As a matter of sport he was trying to jump with others on the sand, two hops and a jump. On the second hop he “fell right down, and couldn't jump at all, and so discovered that his legs were clumsy and ‘dead.’”

Strongly denies syphilis.

The first symptom noticed by the patient in this case was the tabetic pains, six to eight months after the first malarial attack, but it is probable that if the patient had been carefully examined, other evidences of tabes, such as diminished knee-jerks, would have been discovered at an earlier period. The association of the subsequent malarial attacks, with an increase of all the tabetic symptoms, is suggestive and interesting.

Case II., A. S. P.—Although the original fever is said to have been typho-malarial. I give it as the connection between the original form and the secondary nervous disease is quite close.

It is a typical case of tabes, with all the classical symptoms. The disease is now well advanced (lightning pains, gastric crises, anæsthesia and loss of muscular sense of legs, ataxic gait, loss of knee-jerks, Argyll-Robertson pupils, Romberg's symptoms, requires a cane for support, etc.)

In 1862, after a hard march in rain, was taken sick with high fever and diarrhœa; sent to Washington, where he was sick three months; was delirious; was told he had had typho malarial fever, and treated with large doses of quinine. It was six months before he was thoroughly restored to health so as to be able to resume business. About three months from the time he was first taken ill and while convalescing, gastric crises came on. Shortly after this be-

gan to suffer from pains in legs. He now suffers intensely from these gastric crises, which come on about once a month; formerly once in six months.

He also suffers from attacks of the following peculiar character, which are probably modified malaria. He is first taken with severe rigor; this is followed by diarrhœa and profuse sweating. He himself does not think he has fever, though he does not know what his temperature has been by the thermometer. His physicians have always pronounced these attacks (of which he has had eight or ten in the past eight years) to be malaria. Each time he is laid up from two to three weeks and is very sick each time.

No history of syphilis.

Case III., J. A. B.—This case is also one of well-marked tabes:—ataxic gait, static ataxia, tactile anæsthesia and analgesia of legs; knee-jerks lost, ataxia of hands (when writing), tabetic pains in legs and arms and across chest (girdle), pupils somewhat sluggish.

In July, 1863, he was taken with severe malaria and dysentery. The chills and fever were typical in their development. At first the attacks recurred as often as three times a day, then daily, and then every day or two for two months. For two months more he had them frequently, and up to December of that year they recurred at intervals. After the latter date they were replaced by dumb ague; that is, chills without subjective sensations of fever and only occasional sweating. These dumb-ague chills are often very severe. These he has suffered from ever since. In 1863 he was treated with very large doses of quinine. Last winter he had these typical attacks of chills and fever which were very severe, and which, to use the expression of the patient, "put him back just where he was in 1863."

He was discharged in April, 1864. Before his discharge he began to suffer from severe "sciatica," and soon after by severe pains down his arms, particularly right. The sciatic pains in the course of time became transformed into severe burning, boring pains in legs, which have persisted until the present day.

About fifteen years ago he first noticed a tendency to tumble in the dark, and at an earlier period, after getting tired ; numbness of legs was first noticed about twelve years ago.

No history of syphilis.

Case IV., J. F. D.—Typical case of tabes in paralytic stage. The disease is at least of ten years' standing, as he has suffered from typical pains in legs for that length of time. (Examination shows anæsthesia and paralysis of legs, loss of knee-jerks, ataxic gait, loss of muscular sense in feet, Romberg's symptoms, Argyll-Robertson pupils, paralysis of bowel.)

History.—Contracted malaria during the war. Ever since has suffered from severe attacks of chills and fever, having them on an average six times a year.

No history of syphilis.

Case V., A. M.—The morbid process is well advanced. There is present severe tabetic pains, ataxia, loss of knee-jerks, anæsthesia and loss of muscular sense, atrophied optic nerves, color blindness, paralysis, Argyll-Robertson pupils, etc.

History.—In 1863 he had his first attack of malaria. At that time the chills and fever recurred for two months. He has had them from time to time ever since. The chills generally come on in the morning, and the fever follows in the afternoon or evening. He dates what were apparently his first tabetic pains from 1866 ; but the disease did not apparently become pronounced until five or six years ago.

No history of syphilis.

Case VI., E. E. H.—The usual signs of tabes are present. (Knee-jerks absent, Argyll-Robertson pupils, paroxysmal pains in legs, anæsthesia, staggers when walking with eyes shut.) First noticed a difficulty in walking eight years ago.

No history of syphilis.

Has suffered from chills and fever on an average once a

month ever since he had malaria during the war. He also has epileptic attacks.

GROUP II.—DISSEMINATED SCLEROSIS.

Case VII., F. E. S.—The most marked symptom is a fine intention tremor of both hands, particularly noticeable when drinking; hand-writing tremulous; paresis of right hand, which is noticeably weaker than left; slight atrophy of ball of right thumb and first interosseous; voice slightly tremulous; knee-jerks lively, but not abnormally so. He says his memory is poor.

History.—Had chills and fever while in a rebel prison in 1863, and has had them from that time until within a year. Sometimes they are very severe. The first nervous symptoms, in form of weakness of right arm, came on while in prison, after an attack of chills and fever. The tremor he has noticed "ever since he was exchanged from prison, but more especially for the past twenty years."

Case VIII., C. H. S.—The symptoms in this case are pronounced, viz.: moderately coarse intention tremor of hands and head; exaggerated knee-jerks and plantar reflexes; gait unsteady; legs spastic; Romberg's symptom; probably slight anæsthesia of legs (more subjective than objective; speech thick; pupils respond slightly only to light; mentally below par.

History.—Chills and fever while in the army. Has had them frequently ever since. Is laid up, he claims, one-half the time with them. I was unable to learn the exact time at which the nervous symptoms first appeared, excepting that it was at some time subsequent to the first attack of intermittent fever.

Case IX., D. W.—Marked coarse intention tremor of hands and head; drinks with difficulty from a glass; paresis; knee-jerks exaggerated; voice tremulous; ankle clonus.

History.—Had malaria in the army and still has attacks of chills and fever from time to time. Exact period of

development of nervous symptoms unknown but since he first had malaria.

Case X., H. C.—Disease well advanced.

Status præsens. Increased knee-jerks; ankle clonus; intention tremor of hand and head; voice tremulous; possibly very slight loss of sensation in feet; some paresis of hands and legs; Romberg's symptoms; gait ataxic; loss of muscular sense in legs; pupils respond poorly to light; easily tired; slight paresis of bladder; all symptoms marked; memory impaired.

History.—Contracted malaria in army in 1886, and has had attacks of chills and fever from time to time since. Had an attack in 1882, lasting five weeks. Tremor first came on eight or nine years ago, and has had difficulty in walking for four to five years. This patient's memory is poor, and he cannot give a precise account of himself; it is probable that the spinal disease has been of much longer duration than he is aware of.

Case XI., P. M. P.—Fine intention tremor of hands, weakness of limbs, slight ataxia of legs, Romberg's symptom, no anæsthesia; has also acute typical facial paralysis; knee-jerks exaggerated.

History.—Contracted malaria some years ago and still has attacks of chills and fever from time to time. The spinal symptoms followed the first malarial attack (history incomplete).

Case XII.—Dr. Putnam has communicated to me the details of a case to which he kindly allows me to refer. During convalescence from an attack of chills and fever, spinal symptoms developed, and the patient soon showed all the characteristic signs of disseminated sclerosis, which still persist.

Besides these cases I find in my record-book notes of seven others, three of disseminated sclerosis, two of tabes, one of an atypical spinal disease and one of lateral

sclerosis. Although a history of malaria existed in all three cases, my notes are not sufficiently full or explicit to render them valuable as evidence.

I desire, also, to emphasize the fact that these are not all the cases which have come to my notice. Several cases were observed before it seemed to be worth while to note them, and the notes of three other cases lately examined, have been lost owing to a misunderstanding.

I know that there are large gaps in the chain of evidence presented by these cases. It would have been more convincing if in a greater number it could have been shown that the spinal disease developed during the primary fever or immediately after during convalescence. But the length of time that has elapsed since the original attack of malaria in all these cases is so great and the development of disseminated sclerosis and tabes is so insidious that the memory of patients cannot be trusted to establish facts of this kind. Such early symptoms as paralysis of the iris, diminished or increased knee-jerks, slight degrees of ataxia, etc., are of course overlooked by the patients, and even more marked disturbances, such as tremor, slight degrees of anæsthesia, paralysis and neuralgia, either not noticed or ascribed to other than the true cause, as was the case in most of the cases here reported. In case I., for instance, in spite of the long-continued tabetic pains, nothing was thought to be the matter beyond chills and fever, until one day the patient tried to use his legs in an unusual way and found he had no control over them. For these reasons it is difficult to show, even if such be the case, that these chronic spinal diseases develop immediately after a malarial attack. On the other hand, such evidence must not be taken for more than it is worth. The secondary changes which are known to occur in other organs as sequelæ of malaria are not limited in turn to the primary attack. On the contrary, they are more apt to be found where malaria is chronic in the system and are insidious in their development. Parenchymatous nephritis, "amyloid degeneration of the kidney, liver and spleen, hæmorrhagic diathesis, scurvy, tuberculosis, malarial cachexia, etc," etc., may develop gradually in persons who are periodically subject to malaria.

The connection between tabes dorsalis and syphilis is well recognized, though the first symptoms of the former may not make themselves known until long after the primary attack. Now it is a noteworthy fact that syphilis and malaria resemble each other in one particular, namely, they are constitutional diseases in so far as the poison or germs may remain in the system for a long period of time and only show signs of its presence by volcanic-like outbreaks from time to time. In this respect malaria differs from the other fevers, like typhoid, small-pox, etc.; in these, if death does not take place, the poison is eliminated entirely and once for all from the system. Syphilis and malaria, too, may both fairly be said to be amenable to specific drugs, which is not true of the acute fevers. Now in the cases just reported there is one thing common to them all, and that is the patients were all continuously subject to malarial attacks and had been so for years; or, in other words, it is fair to presume that the malarial germs were still in the system. So that these cases are not simply to be looked upon as examples of nervous disease following a single acute attack of fever, such as might follow one of the acute exanthemata, *but rather as cases of nervous disease in persons who are at the same time infected with the malarial poison.*

In looking over the literature of the subject I can find very little in support of this ætiology.

Gowers, Ross, Charcot, Hamilton, Spitzka and Strümpel do not mention malaria in connection with either tabes or disseminated sclerosis. On the other hand, the latter disease has been met with by various observers after other acute febrile diseases, such as small-pox, typhoid, typhus, diphtheria, cholera, etc.; and tabes has been observed to follow typhus, diphtheria, typhoid, acute rheumatism and syphilis.

Erb, following E. Schulze, states that tabes may be caused by a predisposing influence, exerted by previous acute and chronic diseases (typhus, *intermittent fever*, and the like), whether occurring but once or repeatedly.

Tuezek has shown that a true tabes may be caused by the ergot parasite. The fact is that our knowledge of the

effect of poisons upon the nervous system is very fragmentary, and we may find that more diseases than we now imagine are due to unsuspected poisoning, zymotic or chemical, of one kind or another.

Putnam² has shown a remarkable association of different forms of nervous diseases with the previously unsuspected presence of lead in the system, and there is reason to believe that both arsenic and alcohol act upon the central nervous system as well as the peripheral nerves. Tabes has been attributed to absinthe, illuminating gas and other chemical poisons.

As to malaria, it is known that intermmitent paralysis, such as hemiplegia, anæsthesia, neuralgia resembling the crises of tabes, tremor, choreiform spasms, aphonia, insanity, amblyopia, and other disturbances of the nervous system, occur in persons infected with the poison, showing that the latter does have a direct effect upon the nervous system. The symptoms usually occur in the form of what is known as *masked* malarial fever, the attacks taking the place or following the typical chills and fever. These "masked" attacks usually "run their course without fever" (Hertz, in Ziem. Cyclo., vol. II.), or are only accompanied by partial febrile manifestations, as slight chilliness or heat with a rapid pulse or sweating. Hertz thinks they depend on an infection of certain nerve tracts, in the same way as the other portions of the central nervous system are affected in the typical attacks, with resulting chills, heat, dizziness, etc.

Eichhorst (Real-Encyclopædia) says that permanent insanity, paralyzes, anæsthesias, contractures, etc., may occur as sequelæ of malaria.

If further investigations should show that the association of degenerative spinal disease and intermittent fever is more than a coincidence, the anatomical changes that are known to occur in the latter disease will easily render the connection intelligible. Brownish or slate-colored discoloration of the cortical portion of the brain, due to accumulations of pigment matter, has been found in those dying of

² Boston Med. and Surg. Journal, July 28, 1887.

pernicious malaria. According to Hesché, these collections of pigment matter are greatest in the spleen, liver, brain and spinal cord. Hyperæmia, œdema, and numerous punctiform extravasations and softening have been observed also. It would seem *a priori* that inflammatory foci might easily be set up from which secondary degenerations might follow, or the latter might be consecutive to the malarial cachexia, a rather indefinite conception.

I referred in an earlier paragraph of this paper to certain considerations which render the question one of great practical importance. It is this. You are aware that the soldiers in the late war are entitled by law not only to pension for the diseases or injuries contracted during military service, but for the disability resulting from the sequelæ of those diseases or injuries. Now the Government does not, I understand, recognize disseminated sclerosis, or tabes dorsalis, as sequelæ of malaria, and very numerous soldiers have had their pensions disallowed in consequence. If a true causal relation exists, it is apparent that much unintentional injustice has been done, and that it is important in the interests of many who are helpless from spinal disease, that the connection, if a true one, should be established. It would be well if neurologists who have an opportunity to study the effects of malaria and who have not had their attention drawn to cases of this kind would be on the lookout in the future for them.

PERISCOPE.

BY FREDERICK PETERSON, M. D., AND LOUISE FISKE-BRYSON, M. D.,

ATROPHY OF THE DELTOID AND ITS CURE BY PASSIVE CIRCUMDUCTION OF THE LIMB.

G. C. Harrison, writing in the *Lancet*, Feb. 16, 1889, finds the literature of this affection very meagre, the suggestion being generally that it may be a form of rheumatism, which in his opinion it is decidedly not. In studying into the matter he was struck by the peculiar course of the circumflex nerve, which is totally unlike that of any other muscular nerve in the body. His theory of the origin of these deltoid atrophies, then, is that the circumflex nerve, in passing through the quadrilateral space, is rendered liable to injury, and even to strangulation. Falls upon the shoulder, stretching the arm beyond its due limit, or long-continued pressure in certain positions appear to cause atrophy of the muscle, and the writer suggests that the atrophy is due to strangulation of the nerve in the quadrilateral space, varying in degree according to the mode of injury, sometimes causing irritation and consequent hyperæsthesia, and sometimes complete interruption of the nerve-current and paralysis. He relates some eight cases in illustration of his theory and of his treatment by circumduction. His second case is particularly typical, and is here given as an example:

"CASE II.—My next case was a widow lady, who six months previously had been attempting to reach something from a high shelf, when her left arm fell powerless to her side. She suffered much pain, and had been treated medically for six months without benefit before I saw her. The deltoid was quite atrophied; all the other muscles of the arm compared favorably with those of the opposite limb. Circumduction caused considerable pain, and the movements of the humerus were very restricted, owing probably

to the locking of the head in the joint by adhesions or want of elasticity in the teres major and minor ; but I persevered with the treatment, and had the satisfaction of completely restoring the limb, the atrophied muscle gradually resuming its former size and vigor."

LUNACY IN AUSTRALIA.

F. N. Manning discusses some very interesting topics in the *Australasian Med. Gazette*, Jan., 1889. In the provision of reception-houses in New South Wales and Queensland, and of lunacy-wards in public hospitals in Victoria, for the treatment of insanity in its early stages, the Australian statutes are decidedly in advance of those of Great Britain, and, the editor may add, also in advance of those of the United States.

There has been little legislative provision for imbeciles and idiots in Australia, and most of cases find a place of refuge in the ordinary insane asylums, but New South Wales has a separate hospital for idiots. Three of the colonies have institutions for insane criminals.

As regards the criminal insane, he thinks that those who commit crimes while insane should be treated in asylums, but that there should be lunacy-wards in prisons, just as there are sick-wards for those who become insane while undergoing penal servitude, and refers to the successful administration of lunacy-wards in connection with the Scotch prison at Perth, and with the English prison at Woking.

It is interesting to learn from Dr. Manning that the universities of Sydney and Adelaide make the study of psychological medicine a compulsory part of the curriculum for degrees.

The author very sensibly says that no one form of building can meet all the requirements of the insane, and his ideal psychopathic hospital is one that certainly would meet the approval of all who have had experience in the management of large numbers of the insane. He would stipulate for a central hospital for the sick and for acute cases, surrounded by blocks or pavilions of varying form and con-

struction for different classes, and supplemented by cottages for the convalescent, the quiet, and for certain chronic cases. At least a fourth of the total accommodation should be in the form of separate or single rooms. The pavilions should be comparatively small, so as to prevent too large an aggregation of patients.

HOSPITAL TREATMENT OF THE INSANE.

There is certainly very great need of reform in the treatment and management of the insane. Instead of congregating vast numbers of these unfortunates in huge buildings of a cathedral style of architecture, where individuals are lost sight of, where the physician's duties merely become perfunctory, where the chief object seems to be to feed, clothe, and keep patients warm on the lowest possible economical basis, where the superintendent degenerates (with notable exceptions) into a farmer, surveyor, plumber, or architect, a radical change is approaching in the way of greater segregation and individualization.

Dr. H. R. Hopkins, in a discussion before the Buffalo Medical Association, June 4, 1889, upon the above subject, presented ideas so in accord with those of the writer of this abstract, that he cannot refrain from assisting in their diffusion. The most important of these is that of opening general hospitals to the reception of the curable insane, as has been done in Victoria, Australia. The remarks bearing upon this subject are as follows:

"This is the year 1889, and yet it is necessary to call the attention of this society, and of the medical profession which this society represents, to the fact that in the management of the insane we have retained certain demoniacal residua. Our statutes and our usages still provide that before an insane man can be subjected to suitable treatment the machinery of the courts has to be invoked, and he has to be committed to an asylum, to be sure, and yet this asylum is abundantly supplied with locks, bolts, bars, handcuffs, strait-jackets, camisoles, and various methods of treatment hardly compatible with the idea that the insane is only a patient needing medical treatment. Is it not time,

Mr. President and gentlemen, that the medical profession asserted itself and recognized, in fact as well as in theory, that the insane is not demoniac, but only ill? Could anything be more absurd than for a medical man of this day to apply to the courts for permission to treat a case of inflammation of the eye, to place a patient suffering from inflammation of the eyes in that condition where injurious influences are to be excluded from him; or, in case of a fractured femur, to invoke the machinery of the law to enable him to place such restraint upon the patient as experience has demonstrated is necessary to his proper recovery? For myself I can see no lack of similarity between the requirements of one suffering from an inflammation of the eye, or suffering from a fractured femur, and one suffering from mental disease; and it seems to me that it is for the medical profession to assert that it has no conception of an insane man other than as a patient requiring relief, and that the medical profession is in a situation to demand that there shall be no circumlocution, no embarrassments placed between the most enlightened treatment and the suffering citizen in need of such treatment.

"This brings me to a suggestion which I would like to make to the society, and that is, that it is not expedient that the care and management of the insane of any community be placed in the hands of a few individuals of the medical profession. In my judgment the members of the medical profession at large are the safest custodians for this responsibility, and I would suggest for the consideration of this society the propriety of recommending that every hospital have attached to it a suitably prepared ward for the reception of the acutely insane. To these wards, so equipped, patients should be admitted with no more circumlocution than pertains to the admission of patients to the surgical ward, the lying-in ward, or any other department of a well-equipped hospital. If statistics have ever established any fact in medicine, they have established the truth that early treatment in the history of insanity exercises the most hopeful influence over the case."—*Buf. Med. and Surg. Jour.*, Aug., 1889.

INSANITY FROM USE OF GANJA IN BENGAL.

In Bengal, among the predisposing and exciting causes of insanity, ganja takes the place of alcohol. Its effects, though equally violent in many instances, are much more evanescent, and its habitual use does not lead to the same organic tissue-changes. A very large proportion of the graver crimes are committed under its acute influence, but under confinement and deprivation of the drug the mania which it causes rapidly subsides, and leaves the man a quiet and trustworthy inmate, with little tendency to relapse, in the absence of the drug, for the remainder of his life.—*Extract from editorial, "Lancet," Nov. 17, 1888.*

[Ganga, gunjah, bangué, beng, subjee, supposed to be the nepenthe of the ancients, consists of the largest leaves and capsules of cannabis Indica, mixed usually with other substances, like sugar, areca, etc., and is used for chewing and smoking.]

MULTIPLE NEURITIS IN BRASS-WORKERS.

Dr. C. W. Suckling has met with a few cases of ataxia in brass-workers, in one of which the knee-jerks were lost, and the case resembled locomotor ataxia, except that there were no pupil-changes. He considers the ataxia due to a peripheral neuritis, caused by copper-poisoning. He relates the following two cases:

"CASE I.—The patient, a man, aged thirty-nine, has been a brass-worker for twenty years. He has been a steady man, and not addicted to drink. About Easter last he began to suffer from pains in his legs, and especially from aching pains in his calves. The pains in the lower extremities were later on followed by slighter pains in the fingers, and still later by weakness in the legs and hands, so that in a few weeks he was unable to walk. There is a well-marked green line on the teeth, the lower half of the body of each tooth being stained of a deep green color, the typical staining of brass-workers. He complains of his fingers feeling hot, and there is slight tactile anæsthesia in the hands and feet, thermal and painful sensations being

normally perceived. There is marked loss of muscular sense in the legs. He suffers from double wrist-drop and foot-drop. The grasp is very feeble, and the fingers slightly flexed at all joints, there being inability to straighten them, or to extend the wrists. The feet are dropped and flaccid; the knee-jerk is lost on both sides. The calf muscles are tender when grasped, but there is not the extreme tenderness so often met with in alcoholic paralysis. There is emaciation of the arms and legs, but no œdema or change in the nails. The bladder and rectum are unaffected, and there is no bed-sore. There have been no mental symptoms, no dyspepsia or morning sickness, and he has not had syphilis, rheumatism, or gout. He is not phthisical or diabetic, nor has he been much exposed to cold, nor worked in lead. He has considerably improved since his admission into the Queen's Hospital under the influence of iodide of potassium and massage. On admission he was quite unable to stand, and now he can walk about the ward, though his gait is of the high-action type. The electrical reactions in this case were normal, except that the faradic irritability was a little diminished.

“CASE II.—W. G., a man, aged thirty-one, a brass-finisher, was admitted into the Birmingham Workhouse Infirmary in June last. On admission it was found that his gait was ataxic, and that the knee-jerk was lost on both sides. The grasp was very feeble, and he could not fully extend the fingers. The feet were dropped. He complained of numbness in the fingers and toes, and there was some actual anæsthesia in the inner borders of the feet. He had a well-marked green line on the teeth, and had often suffered from attacks of profuse sweating. The pupils responded normally to light and accommodation. There was considerable emaciation of the thighs, legs, and forearms, but no qualitative electrical changes, except that faradic irritability was somewhat diminished. He had been ailing eighteen months before his admission, and had been unable to walk for twelve months. He has greatly improved under the use of massage and the administration of iodide of potassium internally, being now able to walk well without ataxia.”

The author finds relief in these cases to follow administration of iodide of potassium.—*Brit. Med. Jour.*, Dec. 15, 1888.

MASSAGE IN INSANITY.

Dr. G. H. Savage, in a paper on this subject, said that of late there had been too great a tendency to treat all cases of neurosis by massage. This treatment was not only of no use, but was really harmful in some such cases. It might be taken for granted that it was rarely if ever useful in ordinary cases of insanity; in cases of emotional self-consciousness it was bad, both the solitude and the bed being contra-indicated. In hypochondriacal states it was generally harmful, and in most cases of active melancholia it was not useful. Its chief use was in those cases in which the mental depression was associated with physical weakness, loss of flesh, and deficient action of the gastro-intestinal tract. The massage should be continued as long as the general health was feeble, and as long as the appetite was bad. The object was like putting salt in the food given by stomach-tube to create a desire for food, and then to supply it in a suitable form and quantity. He related a specially interesting case of melancholia cured after four years of profound depression.—*Edinb. Med. Jour.*, Sept., 1889.

RAYNAUD'S DISEASE.

Dr. C. P. Combs says in the *Med. Press and Circular*, January 2, 1889, that those who are consulted about particularly bad chilblains during the Winter months will feel interested in watching such cases, as they may turn out to be symmetrical gangrene. It is twenty-eight years since Raynaud described this disease. As an instance, take the following: A girl of sixteen walks in melting snow, and a few days after suffers much pain in her feet. The toes are swollen, sometimes pale in color, sometimes dark red, sometimes dark purple. The pain varies, and is often so intense that the patient will not allow her feet to be touched. The next stage may be that of gangrene, which is usually dry and superficial. In Raynaud's disease we

have the familiar morbid changes of chilblains, the ischæmia of parts inclined to die from cold, the cyanosis of the hot stage of chilblains, the intense pain common to both, and the death of the skin of the worse patches occurs in both cases. It is believed that this condition is due to neuritis in Raynaud's disease, with a consequent vascular disturbance. In chilblains there is probably vascular disturbance only.

The author also compares herpes to Raynaud's disease. The darkened patches of zona, the vesicles, the small sloughs, dry, black, some separating and leaving granulating surfaces, the great systematic disturbance, all resemble the similar changes of the second and third stages of Raynaud's disease.

ON THE TREATMENT OF SEVERE CHOREA BY CHLORAL.

Dr. W. T. Gairdner, writing in the *Lancet*, August 3, 1889, states that in 1870 in treating an unusually intractable and serious case of chorea began using chloral, then a rather new hypnotic. By mistake sixty grains were given to the case, a girl of eight years of age. Symptoms of poisoning set in, and the critical state was not passed until the lapse of five hours. The choreiform movement entirely disappeared and did not return. The girl was discharged recovered in two months. He had continued to treat similar cases in this way ever since the good result obtained in his accidental experiment. Since then many others had found chloral extremely useful for the same purpose. The most recent writer to laud the drug in severe chorea was Dr. Bastian in an article in the *Lancet* of July 13, 1889. In all ordinary cases he should not think of resorting to it, since they readily yield to arsenical or other tonic treatment. But there are unquestionably a few cases in which the tonic regimen and tonic treatment fail, and others in which their action, though ultimately successful, is so slow as to be unsatisfactory; besides a very few in which the disease *per se* is alarming and apparently dangerous from exhaustion, present or proximate, so as fully to justify (not to say excuse) a much more active treatment. In all these

three classes of cases he has habitually employed chloral, and since 1870 chloral *almost alone*. These, of course, have been almost among the worst cases in his experience; and practically these have included *all* the very bad cases. And, therefore, he declares in no single instance has chloral hydrate, with the comparatively modest and limited expectations he has formed of its value, wholly and absolutely disappointed expectation. It has not often *cured*, it is true, directly and at once, as in the case just narrated, but it has always had an important influence in controlling choreic spasm, and has rarely failed in placing the patient *in the way of cure* when followed up by hygienic and other remedies or by lapse of time, which in many such cases is *the* remedy. He recalls several cases of chorea in children so bad that he could not but regard them as having an element of present danger, which have yielded at once as a first step in the treatment to chloral hydrate, under which mainly the cure has been completed. Taking it all round, his experience of this remedy in chorea, while not without qualifications, is certainly not in accordance with the following expression of Dr. Sydney Ringer (*sub voce*): "Chloral *sometimes* restrains the voluntary movement of chorea, *but sometimes it is powerless*." It would be much more correct to say that chloral can almost always be depended on to control the movements of genuine chorea for the time during which its physiological action is fully maintained, but that it is sometimes inadequate to a permanent cure.

URETHAN IN TETANY.

In the *Boston Med. and Surg. Journal*, September 5, 1889, Dr. S. L. Abbott writes that he had a severe case of tenany at the Massachusetts General Hospital, a 'longshoreman, aged forty-three. The disease had lasted six weeks. Having used urethan successfully in severe chorea, he determined to try its efficacy in this disease. Ten grains were prescribed to be given every two hours by day and fifteen at bedtime. After the second day the cramps had disappeared and did not return. Treatment was continued for nine days. He had heard of the patient nine months later, and he had remained in perfect health.

GOWERS (W. R.) ON THE ELABORATE DIAGNOSES OF
NEUROLOGISTS.

A tendency is often to be observed in the present day to underrate diagnosis, or at least the elaborate diagnosis of which the diseases of the nervous system furnish so many examples. In the face of the urgent needs of suffering humanity, with its mute or uttered cry for the relief we cannot always give, our precise distinctions and elaborate processes may seem like an ingenious device for interesting us while the patient suffers. That such an impression is wholly wrong I need not say to those who hear me now; but the tendency is real; it is reflected beyond our own ranks; and I may give one warning—a warning to myself as well as to others,—that we should be always on our guard lest we do anything, by word or manner, that may excite or foster the feeling to which I refer. Diagnosis must come before treatment, but this should make us careful lest we produce an impression that we regard the order of the two in time that also of their importance—an impression easily produced when, as often, the treatment is plain and its methods familiar, while the diagnosis is complex and its processes strange.—*Br. Med. Jour.*, Feb. 2, 1889.

INEBRIETY AMONG THE CULTURED AND EDUCATED
CLASSES.

In an article upon this subject in the *Med. Press and Circular*, January 9, 1889, Dr. James Stewart gave a *résumé* of observations based upon twelve years' experience in the treatment of inebriates. He called special attention to the pathological condition of the cerebral tissue in inebriety, a loss of brain substance as real as the loss of a portion of a finger sliced off accidentally with a knife. Inebriety was a physical disease as clearly marked as many other diseases, and must, to be successfully treated, be dealt with in as scientific a manner as these other maladies. New and sound brain tissue must be built up before a cure could be effected. This required a considerable time, the shortest term being twelve months. Dr. Stewart concluded by stating as his opinion that: (1) Inebriety is a lesion of the

brain which has gone so far as to affect the will-power. (2) Successful treatment based on this pathological dictum must include the absolute cessation of alcoholic drinking. (3) There is no danger in the sudden and complete withdrawal of alcohol if the case, no matter how severe, be in the hands of a skilful physician able to personally direct the hourly treatment from the first. (4) The physician undertaking the charge of such cases ought to be himself a total abstainer, so that moral treatment by example might supplement therapeutic remedies. (5) Permanent recovery need not be hoped for unless both lines of treatment be pursued systematically—during an uninterrupted period of twelve months—in a home from which alcohol is excluded. (6) So-called “cures” effected by bark, strychnine, and other specifics have not proved permanent. (7) The permanence of a cure depends greatly on the after treatment pursued subsequent to the patient leaving the “home.” The family of the inebriate should all become total abstainers, no alcohol being allowed into his or her house except as a drug prescribed by a medical man and dispensed in a medicine bottle.

FALSE NEURASTHENIA.

In the *Prov. Med. Journal*, February 1, 1889, Dr. A. S. Myrtle writes as follows upon this subject:

Here we find symptoms in every respect similar to those of true neurasthenia, and it will take you all your time and patience, as well as tact, to detect the sham from the real. If you hark back a bit, you will find that as a child she showed temper; as she grew, she became fitful, hysterical, and given to the sulks; craved for sympathy, and exhibited little or no sympathy for others. On questioning her, she describes her sufferings in forcible language. She can neither eat nor sleep; has not an atom of strength; suffers from the most dreadful pain, most fearful headaches, and frightful spasms; and should you suggest any portion of her body, from her head to her heels, as possibly exempt from pain, she often resents the insinuation, and declares *that* is the very part where she suffers most. Whilst she

tells you all this in a sort of whine, her features don't show indications of any agony, and, if you watch her, you will find that she overacts her part. Utterly indifferent to the anxiety of parents and friends, or to the trouble and expense she causes, she seemingly finds gratification in watching the unwearied efforts of those around her in doing their best to comfort and help her. Whilst putting on an air of the most abject listlessness whilst you look at or speak to her, if you talk at her you will learn that she has both eyes and ears ; if you assist her in any way she makes herself as helpless as she can—a dead weight. These creatures not only deceive every one around them, but in time they succeed in deceiving themselves. Were it not so, I cannot understand how they continue playing such a sorry game for so long, and with so much strain and fixity of attention to the exclusion of everything else as I have seen them do. If we push our inquiries a little further, we generally discover that there is some obliquity of the moral sense ; an ungratified whim or disappointed affection at the bottom.

INSANITY FOLLOWING SURGICAL OPERATIONS.

In a recent letter to the *Brit. Med. Jour.* (Aug. 31, 1889), Dr. Tait speaks as follows in criticising Dr. E. Denis' book upon this subject :

“I have now performed, so far as I can estimate, between 7,000 and 8,000 operations requiring the use of anæsthetics, and I have had anæsthetics administered in my practice for purposes not involving traumatism probably in 3,000 more instances, and I know of seven cases of sequent—not necessarily consequent—insanity. Of course there may have been others not known to me, and I shall say fourteen cases to cover that margin of error.

“My own practice, therefore, does not yield a proportion of cases of insanity following operations larger than the general proportion of insanity in the adult female population ; and if I include the cases of anæsthesia, it is probably considerably smaller.

“Dr. Denis says: ‘En moyenne, on observe 2.5 cas d’aliénation mentale sur 100 opérations.’ But if this had been the case all of us engaged in active operating practice would have felt the influence of the fact long ago. Personally, I have been struck by the occurrence of insanity after operations as being like the occurrence of tetanus—something to be met with occasionally, but not a matter to calculate upon. If I saw an insanity rate of 2.5 in my operations, it would be more striking than any death-rate in everything but my hysterectomies, and in that class I have already said I have never seen insanity follow in a single instance; and Dr. Bantock’s experience amounts to practically the same result, for his exception cannot really be called one of insanity following an operation. As a *per contra*, I can point to at least thirteen cases where operations have cured insanity.”

LANDRY’S PARALYSIS.

In the *Brit. Med. Jour.*, Nov. 3, 1888, Dr. M. Woodward describes a case of this disease occurring in his practice.

The subject was a laboring man, aged thirty-seven, married, with three children. His wife said, beyond some trembling of the hands, he had been at work in good health. When first seen, with the exception of a feeling of weariness, there was an entire absence of symptoms; on the next visit, two days later, he was sweating profusely, and the hyperidrosis continued until he died. In a few days paralysis of the muscles of the lower extremities was complete, soon those of the trunk and upper extremities were involved; the breathing became difficult, and he died the tenth day. The symptoms followed the course described in standard works, except the sweating, of which there is no mention. The temperature remained normal, or nearly so, throughout, and the muscles flaccid.

INSANITY TREATED BY HYPNOTISM.

This subject came up for animated discussion at the meeting of the British Medical Association in August last. We extract the greater portion of the discussion from the *Brit. Med. Jour.*, Sept. 21, 1889, and reproduce it here.

“Dr. Auguste Voisin, physician to the Salpêtrière, Paris, made a communication on the treatment of insanity and neuroses by hypnotic suggestion, and on the application of the method to the moral and instinctive perversion of backward and imbecile children. He stated that until within the last few years no serious attempt had been made in this direction, and that it was generally supposed that the insane could not be hypnotized. Dr. Voisin had been able to develop this method in his hospital and private practice. Catalepsy ought to be carefully avoided, because the hypnotized individual ought to be able to preserve the use of his senses, especially of hearing. He was convinced that hypnotism was only useful when it was possible to make use of suggestion, and he was firmly of opinion that, as Braid had said, the hypnotic state originated in the nervous system of the hypnotized person. Having described the basis of hypnotic treatment and of suggestive therapeutics, Dr. Voisin detailed the various categories of the insane with regard to whom he had made observations. By this treatment he had cured persons suffering from hallucinations and delusions, and from disturbances of special and general sensation. Suicidal ideas and acute and furious mania had disappeared under the use of this method. Cases of insanity were cited which had only been calmed after several hours. The treatment had also succeeded in the mania and agitation observed during the catamenia. Patients in this category had even remained asleep from six to eight days. The method had also succeeded in dipsomania and in morphinomania. Dr. Voisin had also been fortunate enough to cure obstinate cases of onanism in this way, and had applied the method *à la moralisation des enfants dépravés*. He had thus completely transformed their habits of thought, and had brought them to love the good, whereas formerly they had only loved the evil. He had also succeeded in curing amenorrhœa in the insane, which was a frequent cause of nervous and mental troubles; he particularly insisted upon this point as proving it was possible to influence the functions of the sympathetic system.

“Dr. Yellowlees said his attitude of mind towards this paper was simply one of amazement. Here was something that cured mania, banished hallucinations, cured love of drink and morphine, stopped masturbation, improved the memory, made imbeciles wise, and bad folks good; moreover, it cured amenorrhœa, and the patient menstruated as directed. It resembled nothing so much as the waving of a conjurer’s wand, and saying to disease, ‘Begone.’ Moreover, in nine-tenths of the cases the cure was permanent. If all this were true, their vocation was gone, and they must seek some other profession. Dr. Voisin must not deem them disrespectful if they were a little incredulous as to these wonderful results. It might be that there was something in the vivid Gallic nature that we had not here, or something in Dr. Voisin’s method that they did not know. Certainly they had seen no such results from hypnotism here, and certainly he had never heard of results so amazing as those enumerated in Dr. Voisin’s paper. Nothing would be so satisfactory to the section as to get a demonstration of his method from Dr. Voisin at the Wakefield Asylum next day, and he would earnestly ask for this.

Dr. Langdon Down had witnessed some cases of hypnotism when the patients declared that, under the influence of suggestion, control over the bowels in constipation and over the uterine function could be obtained. Dr. Tuckey had found it successful in the treatment of dipsomania, masturbation, occupation neuroses, such as writers’ cramp, and various neuralgias. The Rev. Arthur Tooth, of Woodside, Croyden, was employing hypnotic suggestion with much success in his institution for inebriates. Out of the first hundred cases he only failed to influence hypnotically twenty-one patients. But his experience in the treatment of melancholia by hypnotism had so far been disappointing. Only six persons out of the hundred were sufficiently deeply influenced to be susceptible to post-hypnotic delusions or suggestion. The common idea, therefore, that the patient was, subsequently to hypnotism, a mere tool in the hands of the operator, was erroneous in the immense majority of

cases. Dr. Ireland thought that perhaps Dr. Voisin had made too sweeping claims for the success of his method of treatment. He himself could not hope that nine-tenths of the patients so treated would derive benefit, but he had no doubt that in some cases much good might result from hypnotism. Braid, of Manchester, had claimed cures quite as startling as those stated by Dr. Voisin. He published a case in which milk was brought into one mamma in a woman, the other remaining flaccid. There were instances of women being violated when in the hypnotic condition. He, however, did not think that because the remedy was capable of abuse it should be proscribed. The same objection might apply to chloroform. Dr. Percy Smith in the past winter had subjected sixteen patients in Bethlem to experiments, with the assistance of Dr. A. T. Myers, who obtained the services of a professional hypnotizer. The following rules were laid down: 1. That any experiments of this sort in the insane should be entirely directed to a therapeutic end, and not merely to elucidate physiology. 2. That the term "mesmerism" should be avoided in speaking on the matter before patients. 3. The patient not to be alone at the time, and, if a female, that a nurse should always be present. He was obliged to state that the results had been almost entirely negative—in fact, he had not been able to get insane patients into the hypnotic state. It seemed almost impossible to secure the necessary attention. He did not think it necessary to fatigue the meeting with details of all the cases, though it was important to publish failure as much as success. He briefly mentioned the methods used, and suggested that, in all probability, patients in France were more susceptible than in England. He thought that the matter should not be put off lightly, and that evidently more experience was needed before a final opinion could be given as to the value of hypnotism in the treatment of insanity.

THE INTERNATIONAL CONGRESS ON HYPNOTISM.

As a valuable addition to the above account of the discussion of hypnotism and insanity, at Leeds, we extract

from the *Lancet*, Aug. 31, 1889, parts of the proceedings of the Congress on Hypnotism, at Paris :

Over a hundred medical men, French and foreign, responded to the summons of the organizing committee. M. Dumontpallier, the president, at the opening sitting, traced rapidly the history of scientific hypnotism, the progress of which was in a large measure due to the researches of the school of the Salpêtrière and of that of Nancy, but the popular knowledge of which in the profession dated back to very recent days only. The study of scientific hypnotism, said the speaker, began in 1876 only, when a commission was nominated by the Biological Society to report on the experiments in metallo-therapy of Burq. The recognition of certain effects produced by metals led the commission to the study of hypnotism, which produced analogous phenomena. M. Dumontpallier, at the Hôpital la Pitié, and M. Charcot, at the Salpêtrière, carried on this inquiry. Then the Nancy school, directed by Liébeault, Bernheim, and Liégeois, worked out one branch or factor in the hypnotism—viz., suggestion—which it made peculiarly its own. Indeed, M. Bernheim endeavors to show that under suggestion may be included nearly all the hypnotic phenomena. It is undoubtedly a powerful element, but many other procedures and physical agents may be used to bring about the hypnotic state independently of suggestion. Thanks to the works, then, of Ladame and others, hypnotism now finds its place in the academies, and no longer meets with opposition from scientific minds. It was still, however, necessary, in order to place this progress on a solid basis, that all experiments should be conducted with scientific reserve and closely criticised, and to consider nothing as a demonstrated fact unless it were confirmed by all or the majority of inquirers.

Such, in *résumé*, was the opening address of the president. The first question which was put before the meeting for consideration was: "The prohibition of all public performances in hypnotism, and the necessity of putting hypnotism under legal control." Finally the proposition was put to the vote and carried; and to it was added the follow-

ing resolution, which was also carried, viz.: "That it is desirable that the study of hypnotism and its therapeutical applications be introduced into the curriculum of medical education."

MM. van Renterghem and van Eeden, of Amsterdam, next communicated the results obtained in the treatment of 414 cases made up of organic diseases of the nervous system, neurotic, mental, and neuralgic cases, etc. The method adopted was the Nancy one of suggestion. In 71 cases there was no result, in 92 slight amelioration, in 98 marked amelioration, and in 104 cases cure; 57 cases were not worked out.

After this the congress proceeded to the discussion of the second question, viz.: "What is the relative value of the different methods of bringing about the hypnotic state and of augmenting suggestion from a therapeutic standpoint?" M. Bernheim, in discussing these points, said that to define hypnotism as induced sleep was not sufficient, for all hypnotized subjects do not sleep. Some have profound sleep, with amnesia on waking; others also sleep deeply, but recollect their hypnotic period; a third category fall into a light sleep only; and, finally, there are others who do not sleep, or believe they do not. The degree of suggestibility varies with each one of these groups, and is very high in the first; the second are also subject to fairly complete suggestion, but in the remaining groups it is incomplete. For example: one can obtain suggestion of movements and sensation, but not hallucinations. A subject may, however, pass from one to another of these categories. The hypnotic condition, then, is not mere sleep, but a peculiar psychical state, the sleep indicating a profound condition of suggestibility only.

F. P.

OPTIC-NERVE ATROPHY PRECEDING GENERAL PARESIS.

The *British Medical Journal*, Sept. 21, 1889, gives Dr. Wigglesworth's statement in regard to optic-nerve atrophy as an occasional early symptom—sometimes the first sign—of general paresis of the insane, and preceding all mental

evidences of the disease. Primary optic-nerve atrophy, for which no cause could be assigned and associated with obscure mental symptoms, might be considered an evidence of approaching general paresis. Dr. Percy Smith cited two cases, at Bethlem, that bore on Dr. Wigglesworth's paper. One, male, aged thirty-five, was blind when admitted, sight having begun to fail three years before admission, with some mental symptoms eighteen months previous to date of entrance in hospital. Thus ocular symptoms preceded mental abnormalities by one year and a half. There was a history of syphilis; knee-jerks exaggerated. Patient eventually died in convulsions. In another case, a woman, the autopsy revealed numerous cortical wastings. Dr. Yellow-lus mentioned a precisely similar instance.

In the same journal are set down Dr. Fletcher Beach's views of the "Causes of Idiocy and Imbecility." His experience, perhaps, might differ slightly from those of others whose patients were drawn from a higher class of society.

1. Hereditary predisposition played such an important part, that Moreau, of Tours, found it present in nine-tenths and the author in seventy-six per cent. of all cases.

2. Next in order came intemperance, combined with other causes (rarely is imbecility due to one cause alone), the parents not considering drunkenness a disgrace.

3. Phthisis.

4. Maternal impressions. Some causes would be found to exert themselves before birth, some at birth, and others not till some time—perhaps years—after birth. Convulsions in infancy, epilepsy, fevers, and injuries he considered more potent causes than others whose experience led them to different conclusions. Tedious labors, consanguinity of parents, and hereditary syphilis—especially consanguinity and syphilis—did not appear to the author to play such an important rôle in ætiology as others had supposed.

TUMORS OF THE PITUITARY BODY.

This subject is considered in the *University Medical Magazine*, Nov., 1889. Tumors in the pituitary body, though

not frequent, have been recorded; cysts have been described by Zenker and Weichselbaum; adenomata have been seen by Ribbert, Weigert, and Weichselbaum; Klebs has recorded instances of sarcoma and carcinoma; Weigert of gummata; and Weichselbaum one case of lipoma. Neoplasms, unless extending beyond the limits of the pituitary body, produce no recognizable symptoms. This was true in five of the thirty-eight cases collected by Rath. In the remaining thirty-three the symptoms indicated the results of cerebral compression—headache, vomiting, giddiness, epileptiform seizures, atrophy of the optic nerve (or papillitis), temporary hemianopsia, hebetude (in thirty per cent.), imperfections of speech and hearing, involvement of the oculo-motor nerve, and of the abducens. In three instances, diabetes mellitus existed, and diabetes insipidus in one. It has been taught that optic neuritis is frequently absent in this form of brain tumor. Rath thinks this is true, because death occurs early in the affection. Bernhardt believes that pressure is too great to admit entrance of fluid into the optic nerve-sheath, and hence a primary atrophy without preceding neuritis. Neuritis may occur late in the disease, supervening upon an already existing atrophy. A curious symptom has been observed in hypertrophy and disease of the hypophysis cerebri. This is a tendency to grow stout. In the remarkable condition first described by Marie, and named by him acromegalia, an acquired hypertrophy of the upper and lower extremities and of the head takes place. Minzowski, in a discussion of this disease, referred to post-mortems that revealed enlargement of the pituitary body.

In a case of suspected tumor of this body, recorded some years ago by de Schweinitz, in addition to other well-marked symptoms, there was excessive sweating, especially of the hands and feet. Rath concludes that when tumors are confined exclusively to this region, there are no diagnostic symptoms. When extending beyond its limits, prominent signs of this disorder are temporal and frontal headache, ocular disturbances, weakness in the legs, and hebetude. A peculiar form of dementia, and of diabetes mellitus or insipidus, would yield confirmatory testimony.

ABSCESS OF THE BRAIN.

In the *New York Medical Journal*, Nov. 9, 1889, Dr. T. M. Markoe reports the following case, which was admitted to New York Hospital, Oct. 2, 1888: A. R., Syrian, aged twenty-two, peddler, found in cellar in stupid condition, unable to give any account of himself. It was ascertained that, a year before the present accident, the man had received an injury to the head. There existed a crucial cicatrix, depressed into thickness of scalp, but not suggesting depression of bone, and situated parallel to and about half an inch behind the fissure of Rolando; cicatrix firm, solid, without sign of inflammation; near centre, a soft spot, as if bone deficient there. Mental condition dull, apathetic; pupils normal, react naturally to light; obliteration of wrinkles of forehead on right side; slight drooping of upper eyelid; some drawing of right corner of mouth; extrusion of tongue normal; right arm without difference in sensation from left, but marked disinclination on patient's part to move it, especially as high as head; sluggishness of movement in right leg, not so marked as right arm. Pulse, 68; respiration, 21; temperature, 100.6°. Patient in condition of active salivation, but does not know for what purpose mercury administered.

Patient kept quiet in bed; thirty grains bromide of sodium and ten grains iodide of potassium given three times a day. Began to be delirious in a mild way two days after arrival.

October 5th.—Convulsion, repeated five times, beginning with twitching of muscles on right side of face, then right arm, right leg, then general. No change in pupils. Attack lasted about five minutes. Next day two more convulsions. Believing that symptoms depended on meningeal inflammation in region of scar of old injury, vesication with blistering collodion was established there, covering space as large as palm of hand. Occasional convulsion afterward.

From October 6 to October 11, temperature remained normal, then rose to 101.6°. Complained of pain in head, restless, sensation of arm and leg on affected side distinctly lessened.

October 12th.—Pulse and temperature increased. Fluctuating tumor in region of vesication, just in front of old cicatrix, extending over frontal and parietal region. Left eye closed by œdema of lids from proximity of swelling. Abscess opened by free incision, through which finger detected bare bone. Release of pus gave relief, and patient seemed brighter.

No real improvement taking place in the man's condition, signs of pressure increasing rather than diminishing, progression of symptoms giving reasonable ground that intracranial suppuration had already taken place, it was decided that an operation afforded the only prospect of relief. This was performed October 13th. Incision already made for evacuation of subcutaneous abscess enlarged, to expose region of original bone injury. Skull found to be deficient over oval space covering more than two square inches. This space filled by firm, thick, fibrous-looking membrane. Appearances were that of skull that had been trephined, and in which the deficiency had been filled by usual fibrous cicatrix. Next step, removal of cicatrix and exposure of brain surface, which was done carefully with scissors. Before half completed, pus oozed and soon flowed freely. Removal of remainder of cicatrix disclosed orifice of cavity extending nearly three inches into substance of brain, the walls of which appeared to consist of unaltered brain-substance. After full evacuation, drainage-tube introduced, flaps approximated by sutures, and antiseptic dressing applied. Improvement followed operation, and there were no convulsions after it.

October 20th.—Pain in head, slight delirium, temperature 105° , pulse 130, pupils contracted. Evidences of paralysis more and more marked, and symptoms of general meningitis. Stupor deepened daily, with 102° to 105° as temperature range, Cheyne-Stokes' respiration, with death, October 25th, twelve days after operation.

Autopsy.—Beneath the flaps made by two incisions, there is an opening through calvaria, from which fungoid mass protrudes. Opening (oval), situated in left parietal bone, has long diameter of one and five-eighths inches; its short-

est being one and one-eighth inches. It extends about one-half inch to left of interparietal suture, and reaches within a few lines of temporal ridge. Anteriorly approaches coronal suture within about a half inch. Precise situation of abscess cavity most important feature of this description. Orifice admits little finger, is surrounded (and in fact formed) by mass of granulation-substance, which projects from surface of hemisphere about one inch, more anteriorly than posteriorly. Meninges consolidated, adherent to brain surface all round orifice of cavity, most extensively on front boundary. No evidence of general meningeal inflammation. Topography of abscess cavity brings it anterior to fissure of Rolando, which its posterior border skirts closely for about an inch. It approaches longitudinal fissure within perhaps four lines; anteriorly occupies posterior part of second and portion of first frontal convolution, reaching downward to, perhaps encroaching on, the third frontal convolution. Main development of abscess, therefore, in the middle portion of ascending frontal convolution.

The case illustrates the general law (von Bergmann), that abscess of substance of cerebral lobes, except metastatic and tubercular abscesses, is never primary or idiopathic, but depends upon traumatism, upon some focus of suppuration outside of brain-substance. In this instance, abscess undoubtedly resulted from double trauma: first, an old compound fracture with its resultant cicatrix; and, second, the recent injury caused by fall into the cellar. It is well to note that absence of high temperature does not necessarily exclude diagnosis of cerebral abscess. Pulse also, during whole observation, was not materially accelerated. Just before death the rise of temperature to 105° and the pulse to 130 seemed to indicate general meningitis, which the post-mortem did not verify.

The case is also interesting from the standpoint of cerebral localization. Clinical symptoms pointed to seat of lesion, but at no time was paresis strongly marked. All experimentalists on localization seem to agree in stating that centres for face, arm, and leg lie in the convolutions on *each* side of the fissure of Rolando, and not on one side

or the other alone, which may explain the slight symptoms during life as far as paralysis is concerned.

HYPNOTISM IN THERAPEUTICS.

The *Medical News*, Nov. 2, 1889, contains an editorial with this title, that offers suggestions and criticisms of value. Being in a transitional stage, the future of hypnotism is as yet difficult to prophesy. Until the time of Braid, intentional imposture seemed the only rational explanation of mystical theories and absurd statements. Braid undertook the study of the subject in a spirit approaching the scientific, concluding that this curious psychological condition could be explained without the aid of magnetic fluid or mysterious force. Esdail, a contemporary of Braid, utilized the hypnotic state as an anæsthetic, performing more than six hundred surgical operations on persons under its influence. Little credence was at first given to Esdail's statements; but a committee appointed by the Indian government substantiated them in every particular.

Systematic use of suggestion during hypnosis began with the so-called Nancy school, who, represented by Bernheim and Liebault, claim to cure all manner of ailments, to elevate depraved characters and exorcise vicious propensities. Charcot, while adding much to the knowledge of hypnotic phenomena, has studied its curative properties less than Voisin and others of the Salpêtrière. Voisin has obtained cures of hysteria, hystero-epilepsy, and probably of dipsomania by means of hypnotic suggestion. Among the insane he claims to quiet mania, remove hallucinations, produce natural sleep, and control obstreperous patients. His assertion, that ninety per cent. of the mentally diseased are susceptible to hypnosis, is open to criticism. The dominant influence of a strong intellect over a weaker is not to be forgotten, and the skillful guidance of the imagination is in itself a curative agent. Torel disagrees with Voisin as to the wide applicability of hypnotism in the treatment of insanity.

Corval (*Therapeutische Monatshefte*, Sept., 1889) publishes a long list of diseases where he has seen suggestive

therapeutics produce remarkable results. Could his statements be unreservedly accepted, there would now exist a specific for acute alcoholism, every variety of neuralgia, lightning pains of tabes, epilepsy, deafness, tinnitus aurium, asthma, and muscular rheumatism. It is not affirmed that relapses can be prevented, but Corval thinks them less frequent than with ordinary methods of treatment. In spite of appearances, Corval's deductions are dispassionate, and to this effect: In suggestive therapeutics we possess a mode of treatment that is sometimes curative, sometimes palliative; judiciously selecting cases and avoiding unnecessary experiment, this measure is no more dangerous than many other curative means; its indications and contra-indications being as yet imperfectly defined, its use should be confined to cases in which the usual treatment is unsatisfactory or dangerous—replacing morphine as an anti-algesic and chloroform as an anæsthetic; the method should be studied earnestly and scientifically; and, to prevent abuse and disaster, its practice should be confined, by law, to physicians.

It is extremely doubtful if the use of hypnotism ever becomes general. It must not be forgotten that it is sometimes the reverse of harmless. Epilepsy has grown more severe, hysteria has been produced by it in subjects previously well, and permanent hysterical contractures have developed. In certain instances it has been impossible to arouse patients from the hypnotic sleep for weeks.

TREATMENT OF "NERVOUSNESS."

The Cincinnati *Lancet-Clinic*, of Nov. 2, 1889, contains a paper with the above title, by Philip Zenner, A.M., M.D. In this condition there is either inherited or acquired predisposition. Cause for the outbreak consists in the circumstance or mode of life, in special systemic conditions, or in local pathological processes which act as reflex excitants. Indications in treatment are: Building and toning up of nervous system, removal of causes of disease whenever possible, together with whatever moral influences that can be brought to bear upon the patient. The treatment begins

with the first examination, the physician's success depending largely upon his mode of address, thoroughness, and patience at that time.

In all these cases there exists the basis of real disease. To tell a patient conscious of his own suffering that nothing is the matter with him, causes him to exaggerate the nature of his trouble and to doubt the physician's skill. To say that the disease is functional, and therefore curable, combines both truth and tact, and reassures the patient. Confidence and hope are two powerful therapeutic agents. The cause or causes of the condition for which the sufferer seeks relief must be looked for and removed whenever possible. Overwork, anxiety, grief, disappointment, and other sources of emotional storm, may be discovered readily enough. They are not so easy of removal. Bodily ailments respond more easily to remedies. Any exhausting disease may be followed by nervous conditions. Syphilis, gout, lithæmia, or malaria sometimes appear to be direct or indirect causes, and furnish an indication for treatment. Local pathological processes, in a reflex way, appear to excite or maintain nervous symptoms. The most common of these are: obstruction of the nasal passages, eye-strain, stomach trouble, disease of the uterus and of the prostatic portion of the urethra. Such diseases should be sought out and remedied, though it must not be forgotten that local processes like these only produce general disease when the soil is favorable for it.

The general treatment of the nervous system is always essential, usually more so than the treatment of the local trouble, for the nervous disease often disappears while the local trouble remains. On the other hand, the latter may be cured without favorably affecting the nervous manifestations.

Hygiene—the proper regulation of the manner of living—is the most important agency in giving tone to the nervous system and removing nervous symptoms. Sleep, diet, out-door exercise, and adequate employment and recreation are the physician's affair. Hot water, where there is lack of appetite, is often of value. Where there has been over-

work, or strain from worry, great responsibility, etc., rest should be ordered. The kind of rest is of moment. A change of activity is often greater rest, both to mind and body, than doing nothing. Inactivity too often favors introspection and brooding, which are to be guarded against as much as possible. Thought must be directed into healthy channels, away from the individual himself, until the proper relations to others are normally adjusted.

The first in the list of special therapeutic measures is hydro-therapy, comprising hot, cold, and temperate baths, medicated and mud baths, wet packs, douches, sponge-baths, etc. Personal trial determines the kind best suited to the case. The simplest form, the sponge-bath, can be administered at home. Hydro-therapy exerts a beneficial influence in nervous cases, partly as a calming agent, quieting the patient, inducing sleep, and relieving pain. Its influence in increasing the heart's action, hastening the circulation, and promoting tissue-changes is probably greater than its soothing properties.

A therapeutic measure of manifold application is electricity. The faradic current is often serviceable. General faradization acts as a tonic, improving the condition of the whole nervous system. Local faradism assists in relieving pain and spasm. The galvanic current, though less commonly employed, has a wider range of usefulness. Central galvanism exerts a general tonic influence, like general faradization. A mild current to the head produces good results, favoring normal sleep, and quieting and giving tone to the nervous system. Static electricity probably has a more limited range of usefulness than the other forms, but it often affords much aid in the management of "nervous" cases. The general application of sparks all over the body is an invigorating process, the static head-bath is calming and favorable to sleep, while more limited applications tend to relieve pain, spasm, and the like.

The moral influence of the "big machine" is not to be ignored, patients expressing themselves as great believers in its efficacy. Drugs form the least important element in treatment, and often the patient is better off without medi-

cines. Tonics are most frequently indicated, as quinine, arsenic, strychnia, cod-liver oil, etc. Remedies for the mere relief of symptoms—sleeplessness, excitability—are best omitted altogether. The most valuable of this type are the bromides. Given systematically for a short time, they put the patient in a better condition temporarily, thus allowing other measures to bring about a cure. Severe cases that induce chronic invalidism are best treated by the Weir Mitchell plan—rest, massage, electricity, and excessive feeding—when there is loss of flesh and impoverished blood. Change of scene, mountain air, ocean voyages, sea-baths, etc., are powerful adjuvants.

Success in the treatment of functional nervous disease depends largely upon the physician himself; on the confidence he inspires; on the diagnosis; on the appreciation of other ailments that have to do with the development of symptoms, and the management of such ailments; on the proper estimate of each individual case, from the moral, mental, and physical standpoint, in order to direct the amount and kind of work, to encourage, uplift, and inspire, and to manage wisely the special therapeutic agencies employed. As the physician understands and does these things well, so his success will be. Failure in methods and appreciation means failure to cure the patient.

ANTIFEBRIN IN EPILEPSY.

From the employment of antifebrin in epilepsy, Th. Diller reports, in the *Therapeutic Gazette*, June, 1889, these results from the administration of three or four grains, three times a day: A reduction in the number of fits a month, ranging from twenty-five to seventy-five per cent., as compared with months when tonics or bromides were taken; remedy well borne without apparent mental or physical depression; no skin eruption; in cases where attacks are very frequent and immediate control desirable, the bromides are far more valuable than antifebrin.

PEROSMIC ACID IN EPILEPSY.

In the *Therapeutic Gazette*, Nov. 15, 1889, are Dr. Carl Schroeder's statements concerning the effects of perosmic

acid on eight epileptics, to whom were administered one-fifth of a grain daily in pill-form. Two were somewhat improved, while in the other six no action whatever was perceptible. The experimenter suggests that, if treatment had been prolonged, other results might have been obtained, as elsewhere the use of this drug has proved more satisfactory.

BRAIN SPECIMENS, CHIEFLY ILLUSTRATING LOCALIZATION.

The *University Medical Magazine*, Nov., 1889, contains Dr. Charles K. Mills' paper upon this subject. The brains examined were those of persons manifesting widely different conditions, as oro-lingual monoplegia, hemiplegia, blindness, deaf-mutism, etc.

In the same journal Dr. J. H. Musser's article, on "Some Clinical Aspects of Vomiting, gives the mechanism of this act, its causes and varieties, and its value as a symptom when existing in conjunction with others. Habit vomiting may be due to neurasthenia or hysteria, or to some distant irritation. Sudden painless vomiting in the aged is always of grave omen. The first set of causes of vomiting are those which stimulate the centre directly, independent of organic disease of the brain, as toxæmia, initial stage of fevers, ethers, alcohols, alkaloids, gases, etc.; the second, causes acting centrally in one sense, associated with organic disease about the centre or its vicinity, as cerebral tumor, abscess, inflammation; the third set of causes are organic or functional disturbances of the organs of special sense, as taste, smell; and, fourth, impulses transmitted from various organs of the body by their associate efferent nerves. The author mentions a potent and frequently unrecognized cause of vomiting—drugs given for the relief of abnormal conditions.

TREATMENT OF PSYCHOSES BY OPIUM.

The *Buffalo Medical and Surgical Journal*, Oct., 1889, quoting from the *Therapeutische Monatsheft*, gives the results obtained by Dr. Ziehen, of Jena, through the admin-

istration of opium in mental disease. It was given to ninety-seven patients. Forty three suffered from melancholia, four from mania, and fifty from paranoia. Seventy-nine per cent. (thirty-one persons) of the cases of melancholia recovered. The value of opium in senile melancholia was particularly marked. In mania the drug was of no avail. The bromides and hyoscin were preferable. In paranoia without hallucinations, opium is contra-indicated. It was found efficacious wherever paranoia hallucinatoria existed, twenty-four out of twenty-eight cases recovering.

INSULAR SCLEROSIS OF THE BRAIN.

The *Medical News*, Nov. 16, 1889, contains a clinical lecture with this title, by Dr. Henry M. Lyman. Patient, aged thirty-four, canvasser, much exposed to the sun, drinks beer, has had rheumatism, reports a stroke, but no illness previous to the attack, except some nervousness, the attack coming on while he was quietly sitting. For fifteen minutes he could not speak, had cramps in both hands, but did not fall or lose consciousness. The trouble has been growing worse. Reflexes, heart-sounds and vision normal. The right arm is getting weaker and has numb feelings lasting half an hour at a time. No syphilis. Syphilitic disease involves the arteries of the brain; rupture of a blood-vessel is therefore common. Rheumatism predisposes to cerebral difficulties. Minute fragments of vegetations may be broken off from the valves of the heart and washed into the circulation, obstructing the blood-vessels in a certain territory of the brain—as the Sylvian artery—and thus producing the symptoms of a stroke. A rupture or plugging of an artery results in arrested functional activity. When the left side of the brain in the region of the Sylvian artery is affected, the power of speech is interfered with; when the right side, there is no loss of speech, but paralysis of the limbs. The patient cannot hold a glass of water steadily, yet there is not so much agitation of the hand as in brain-tumor. The left hand is steadier than the right. In writing an unfamiliar sentence, there is an irregular, angular character to all the strokes, which present characteristic inequality. This tre-

mor is often seen in old age, for advancing years induces atrophy of the brain and consequent tremor of the hands. Narcotic poisons (as tobacco), loss of sleep, and excessive venery will do the same; but the most serious cause is disease of the brain substance itself, either a sclerosis in disseminated patches, or injury to the brain-substance, here and there, consequent upon disease of the cerebral blood-vessels, which seems to be the condition in this patient. In sclerotic disease invading the brain there is a tendency to occasional paroxysms of paresis, perhaps of epileptiform attacks, which are multiple in their development. Sometimes the patient becomes completely paralyzed and comatose, consciousness and speech returning, and, in a few days, the power of locomotion. The diseased condition reappears after a time. Chronic inflammation of the cortex, which constitutes the foundation of parietic dementia, frequently produces such conditions; so, too, does chronic disease of the blood-vessels, as in the present case. Together with the tremor after the attack, there is an irregular utterance of speech, each syllable being uttered with special effort in an unnatural and artificial way, owing to the lack of a continuous and equable flow of energy from the brain. There is spastic rigidity of the muscles on the right side, and the tongue trembles when protruded.

Biniodide of mercury, about a twenty-fifth of a grain three times a day, frequently brings about considerable improvement. It may be given for months without danger of salivation.

ACROMEGALY.

Brain, for July, 1889, contains Pierre Marie's observations on this strange disease, in which the most prominent symptom is a striking non-congenital hypertrophy of the extremities (hands, feet, head), and therefore named by the author, in 1885, acromegaly (from *ακρον*, extremity, and *μεγας*, large). The hands are enormous, like battledores; fingers, sausage-shaped; nails, flattened, widened, but short, and striated longitudinally, as well as sometimes curved upward when the palm is outstretched on a table. The arm, except

the lower part, maintains its usual size. The feet are huge, with an enormous pad of tissue on the external border; malleoli, head of fibula, and upper extremity of tibia are also increased in size; otherwise the leg does not greatly exceed the normal. The knees often appear prominent in consequence of enlargement of the patella and the condyles of the femur. The diameter of the thigh is unchanged. The cephalic extremity presents an increase in bulk, most marked in prominent parts of the face, as the eyelids, nose, cheek-bones, and chin. The chin projects downward and forward, the lower jaw is increased in size, the teeth being separated in consequence. The tongue is of enormous dimensions, its volume sometimes double the normal, but the shape also perfectly regular. The ears vary, sometimes being notably increased and often remaining unchanged. All the tissues undergo more or less marked alteration. The vertebræ are much hypertrophied. There is kyphosis, a certain degree of scoliosis and lardosis. While the thyroid may be slightly hypertrophied, it is never absent. The thorax appears flattened laterally, and prominent antero-posteriorly. The sternal region protrudes obliquely from above downward, and from behind forward, the xiphoid appendix being enormous, its free extremity projecting above the level of the sternum. Respiration seems to be especially diaphragmatic. There is a somewhat massive appearance of the pelvis. The joints are rather thick, sometimes nodose, often the seat of cracklings and pains more or less acute. The muscles, except in the cachetic state, are thoroughly well developed, muscular strength sometimes being above the average. Headache is present in the greater number of instances, sometimes of a severe character. Sight is often and most manifestly affected.

In an advanced stage there is complete blindness, due to compression of the optic nerves by the enlargement of the pituitary body. Even when there is slight visual trouble, the ophthalmoscope reveals indications of optic neuritis. Hearing may be equally affected. The skin is generally flaccid, sometimes dry, most frequently presenting a yellow-brown discoloration, or a slightly olive hue, most marked

on the eyelids; there may be vergetures, one case presenting a few pendulous growths of molluscum. The hair and beard are always thick and coarse. The increased size of the larynx may account for the depth and strength of voice. Some patients have an almost insatiable appetite and excessive thirst. Diabetes is a frequent accompaniment of acromegaly.

There is an increase in the size of the heart, and a tendency to venous dilatations (varicose veins, hæmorrhoids). In women there is suppression of the menses, an early phenomenon, from which the commencement of the disease may be dated. Psychical functions are most often well preserved—the good humor of the patients often contrasting grimly with their miserable condition. On the other hand, intense melancholy may drive them to suicide.

The course of acromegaly is of long duration—ten, twenty, thirty years and more. Its onset seems to occur between the ages of twenty and twenty-six. Concerning this point there is not sufficient data. Confinement to bed precedes death by a few years, which, when it comes, is unexpected, with indications of syncope. Acromegaly may possibly be confounded with Virchow's *leontiasis ossea*, *elephantias*, *myxædema*, and the *maladie osseuse de Paget*. Certain forms of *rachitis* also present an appearance similar to that of acromegaly; also *macrosumia* and *Friedreich's disease*. A careful study of the history of each case prevents confusion on this point.

OVER-STRAIN AND UNDER-POWER OF BRAIN.

The *Alienist and Neurologist*, by Dr. C. H. Hughes, of St. Louis, calls attention to the fearful demands that modern life makes upon the nervous system. The electric light, the telephone, the ticker, the phonograph, cable-cars, elevated trains, fifteen minutes for refreshments, amusements and recreation at hours best devoted to sleep, together with stimulation in the form of tea, coffee, or alcohol, certainly constitute a state of things. We sleep on the go and go in our sleep, and consequently mind is a poor affair without a strong brain and body to support it. Duty de-

mands that we save ourselves for our brains, and save our brains for our mind's sake, instead of galloping through life and trotting into the grave. The voice crying in the wilderness is occasionally of avail, especially when it is a scientific voice. Dr. Hughes's paper is interesting and most timely, and based upon the idea that, whatever the real nature of mind may be, it is so intimately allied to organism as to be practically inseparable from it during life.

HYSTERICAL ANÆSTHESIA, WITH A STUDY OF THE FIELDS OF VISION.

In the *American Journal of Medical Sciences*, for Nov., 1889, a paper by John K. Mitchell, M.D., and G. E. de Schweinitz, M.D., treats of this subject. Galczowski was the first to study acromatopsia or dyschromatopsia in hysteria, especially in cases where hemianæsthesia is present. Amblyopia sometimes accompanies hysterical losses of sensibility, with changes in the extent or arrangement of fields of color-vision, or total loss of color-perception in one eye. Physiologically, the field for blue is the largest; yellow, orange, red, green, follow in this order; and, last, violet is perceived only by the most central portions of the retina. In pathological states the characteristic conditions appear in some sort exaggerated, but in varying degree. The various circles narrow concentrically after a fashion more or less marked, following the rule established by the normal condition (Charcot). But some of the French patients examined were found to have a reversed order of color-perception instead of simple concentric diminution of color-fields; and in some bad cases there was total loss of color-sense, everything appearing like a sketch in India-ink. Bernutz states that hysterical anæsthesia is limited to one part, perhaps of the skin, perhaps of the mucous surface, especially the conjunctiva of the left eye.

The authors give an interesting case of an over-indulged girl of sixteen, the youngest of a large family, and small for her age, who menstruated irregularly without pain, and became ill in consequence of being sent away to school contrary to her inclination. There she developed aphonia, and

what Weir Mitchell calls "hysterical ataxia," refusing food, and screaming if any noise offended her hearing, which was extremely sensitive. The fear of fall or accident from incoordination induced a decided paresis of the will. She was analgesic from head to foot, there being no sensation in the breast or under the finger-nails. To the use of two faradic brushes on the nipple—the most severe test—the patient was perfectly indifferent. The sense of touch was fair, localization good, appreciation of thermal differences very feeble.

The examination of the eyes revealed the following conditions: Vision one-half and two-thirds respectively of normal, which, by the correction of a hypermetropic astigmatism under a mydriatic, rose to full sharpness of sight. The following formula was found to correct the optical error:

$$\text{O. D.} + 1.00^{\circ} \text{C} + .90 \text{ axis V. } \overset{20}{\text{—}}.$$

xx

$$\text{O. S.} + 1.50^{\circ} \text{C} + 1.^{\circ} \text{axis V. } \overset{20}{\text{—}}.$$

xx

There was deficiency in the amplitude of accommodation corresponding to the error of refraction, together with a low degree of insufficiency of the internal recti. The ophthalmoscope revealed oval disks, somewhat distended and slightly tortuous retinal veins, with undue prominence of the central lymph sheaths. There were no abnormal pupillary phenomena. The form-fields in each eye were absolutely normal. The color-fields—blue, yellow, red, green—followed in the order given and without any material contraction. Thus in the right eye:

	Outward.	Upward.	Inward.	Downward.
Blue . .	75	38	42	48
Yellow . .	75	28	32	38
Red . .	55	30	25	35
Green . .	45	20	25	25

The left eye presented no material difference from that just recorded. These observations were several times repeated, the last time just before the patient was discharged.

Proper treatment brought about a complete cure in three months.

The authors cite eight cases, selected at random because of changes in sensibility, together with alteration in the visual field, and find that the only conclusions possible to formulate are :—that general anæsthesia, infrequent though it be, cannot for this country be supposed so rare as in Europe. On the other hand, complete or nearly complete unilateral anæsthesia, while commonly present, is by no means so constant a feature of ordinary hysteria with us as with foreign observers. Either of these states as a symptom is far less often accompanied by changes in color-perception than French and German writers would lead us to expect, the changes being practically absent in cases of complete anæsthesia. Foreign writers see more often than Americans grave hysteria with convulsive seizures, hysteria in the male and hystero epilepsy ; so also they find hemianæsthesia with achromatopsia more common. The frequency of concentric contraction of the fields in such states is as true for this country as for Europe.

BUTYLCHLORAL IN FACIAL NEURALGIA.

The *Medical News*, Nov. 9, 1889, calls attention to Liebreich's recommendation of butylchloral in facial neuralgia, in doses varying from fifteen to forty-five grains a day. The formula is as follows :

Butylchloral,	-	-	-	-	45 to 75 grains.
Alcohol,	-	-	-	-	2½ fluidrachms.
Glycerine,	-	-	-	-	5 fluidrachms.
Distilled water,	-	-	-	-	4 fluidounces.

Of this the patient is to take a tablespoonful at a dose.

PREVENTION OF ATTACKS OF MIGRAINE.

The *Medical and Surgical Reporter*, Nov. 2, 1889, quoting from the *Allgemeine med. Central-Zeitung*, No. 39, gives Dr. Hammerslag as authority for the following combination of remedies for preventing attacks of migraine, which he states has not failed him :

R

Caffeine citrat,	-	-	-	-	-	gr. xv.
Phenacetin, -	-	-	-	-	-	gr. xxx.
Sacch. albi, -	-	-	-	-	-	gr. xv.

M. Fiat pulv. Dis. in capsulas No. x.

One capsule may be taken, in the intervals of the attack, every two or three hours. Phenacetin does not act so promptly when given alone.

HYSTERICAL BABIES.

The Cincinnati *Lancet Clinic*, Nov. 9, 1889, quoting from the *Medical News*, gives the following concerning infantile hysteria : The view that hysteria is a pathological condition of the gray substance of the brain and therefore a physical disease, not a neurosis, is shared by Liebermeister, Hagenbach, Burkardt and Duboisin. These maintain that physical symptoms are never absent, and in some cases are the only ones that exist. Hysteria in children is of the simplest type ; and for this reason such cases are especially adapted for the study of the disease. Twenty-four cases of infantile hysteria, studied by Burckardt and Duboisin, presented evidences of predisposition to the disorder. In 58 per cent. a hereditary neuropathic tendency could be traced, while in 50 per cent. there was hereditary predisposition to tuberculosis. Three light cases were free from these tendencies. All the patients, except two, were anæmic. Two had suffered from poliomyelitis anterior acuta. Eight cases were caused by fear, shock, etc. Few instances of ultimate cure were observed. The majority remained anæmic, continuing to be troubled with either headache, palpitation, nervousness, bodily and mental weakness, weakmindedness, or hysterical psychosis. Prognosis may therefore be regarded as unfavorable.

L. F. B.

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

THE REPORT OF THE STEVENS COMMISSION.

The commission appointed about two and a half years ago, by the New York Neurological Society, to investigate the methods pursued by Dr. George T. Stevens, in correction of ocular defects for cure of various nervous affections, made its report on Tuesday, November 5th. The commission consisted of Dr. Edward C. Seguin (chairman), Dr. M. Allen Starr (secretary), Dr. C. L. Dana, Dr. W. R. Birdsall, Dr. David Webster, Dr. Oliver Moore, and Dr. Frank Foster. The commission confined its work to the consideration of chorea and epilepsy.

The report, to say the least, was not flattering, in spite of a number of decided cases of improvement. Dr. Stevens protested against the commission's report as premature, and held that the investigation was improperly conducted and the results had not been fairly recorded in the report.

The discussion was postponed to the next meeting, December 3d.

A stated meeting of the New York Neurological Society will be held on Tuesday evening, December 3, 1889, at 12 West Thirty-first Street, at eight o'clock.

Order: 1. Presentation of a case of congenital bilateral pleuroplegia (abducens paralysis) with facial paralysis, by A. Schapringer, M.D. 2. Discussion on the report of the commission on the treatment of epilepsy and chorea by the correction of ocular defects. 3. A case of cervical paraplegia from dislocation, presentation of cord, by Christian C. Herter, M.D. 4. Neurotic tumors of the breast, by E. P. Fowler, M.D. George W. Jacoby, M.D., president; Frederick Peterson, M.D., 201 West Fifty-fourth Street, secretary.

AMERICAN NEUROLOGICAL ASSOCIATION TRANSACTIONS.

Dr. MORTON PRINCE, of Boston, presented a paper on
MALARIA AS A CAUSE OF DEGENERATIVE DISEASES OF THE
SPINAL CORD. (*See page 585.*)

Dr. N. E. BRILL said that his attention had never before been directed to the possibility of such a relation, but he

thought the details presented were rather too meagre for an absolutely correct decision to be made in the matter. As neuritis and malaria were frequently associated, possibly some of these spinal cases might be coincident in the same manner. Both malarial and cord disorders are very common.

Dr. L. C. GRAY saw no reason why the malarial poison should not cause spinal diseases. We do not yet know the exact character of the poison. But there is a sort of periodicity in many spinal and cerebral disorders, as well as in peripheral nervous affections, which is often as marked as that of fever and ague. He had noticed this particularly in intracranial syphilis. Some of these neuropathies were even relieved by quinine, as he had observed in cases of tabes. When we speak of latent malaria and rest our diagnosis merely upon periodicity, there is danger of error. If Laveran and Councilman were right in their discovery of the malarial germ, this should be looked for in such cases as the author had described in order to corroborate with certainty the diagnosis. Improvement or recovery under the use of quinine was no criterion.

Dr. E. C. SPITZKA saw nothing in the nature of the case to forbid an etiological association between malaria and cord disorders, but he agreed with the preceding speakers that sufficient proof of such relation had not been adduced. To show an etiological significance, it was necessary to present absolute and intrinsic proof of it. If it could be shown that, as in syphilis, there were, at the time of the malarial attack, nervous symptoms, it would be a different matter. In the secondary fever of syphilis there was absence of the knee-jerk. Most nervous symptoms in malaria were of a neuralgic character. The anæsthesias differ from those of tabes. It had been found that pigmentary thrombosis and embolism were the most frequent causes of serious nervous disturbances in malaria. He believed, however, that, like any other cachexia, antecedent malaria might predispose to spinal affections. He had observed a case of parietic dementia of the tabetic type which was probably influenced etiologically by severe malaria.

Dr. H. M. LYMAN, of Chicago, could recall but two cases where it had been intimated to him that the origin of tabes had been malarial. In one he traced the actual cause to a subacute spinal meningitis coming on after exposure to wet and cold. The other case, instead of being true tabes, had proved to be a rheumatic neuritis. He had in his experience seen nothing to support the idea of a malarial etiology in tabes. A complete and perfect history, together with a microscopical examination of the blood, should be required for an indisputable diagnosis of malaria.

THE PRESIDENT could not recall a single case of tabes or multiple sclerosis which presented any relation to malaria.

Dr. PRINCE thought there could be no question as to the correctness of diagnosis as regarded the nervous symptoms in his cases; and as to the antecedent malaria, all of his cases were men who had been in army service, and he had documentary proof of their having suffered from that poison, in the shape of Government records. The really doubtful question was whether the malarial poison still existed in the system at the time of the development of spinal disorders. In some of them there was evidence of its persistence in the shape of typical malarial rigor and pyrexia. He did not consider his etiological explanation decisive, but merely suggestive.

Dr. SPITZKA thought that there were many cases of nervous disease owing their origin to exposure during the war which were contemptuously rejected by the Pension Bureau. It seemed to him that the Association ought to call the attention of the Government to the great injustice which might be done.

THE PRESIDENT, although once an army surgeon, and therefore prejudiced in favor of the soldiers, had been, on the contrary, struck by the vast number of fraudulent nervous cases which were awarded pensions.

Dr. W. M. Leszynsky, of New York, was then elected to active membership, and Mr. Victor Horsley and Dr. David Ferrier, of London, were elected to honorary membership. The resignation of Dr. A. D. Rockwell, of New York, was accepted.

Asylum Notes.

MATTHEW D. FIELD, M.D.

P. O. Hooper, M. D., superintendent of the Arkansas State Lunatic Asylum, Little Rock, Ark., writes :

“ I do not know that there is anything very distinctive in the methods of treatment in use in this institution. My experience has not led me to place great confidence in the direct curative power of drugs in any of the psychoses. In acute cases, of whatever sort, my endeavor is to sustain and improve the physical health as far as possible by nutritious diet, exercise, and the treatment of local or general disease that may be found to exist, and to remove, as far as possible, any occasion of mental excitement or distress. Under such treatment the large majority of those who are received during the earlier stages of their mental derangement recover, temporarily at least, although, unfortunately, relapses are, in my patients, as I judge is the case with others, far from rare. In paroxysmal cases I have not succeeded in finding any means either of preventing or cutting short the attacks. In chronic cases much can be accomplished by employment, recreation, and persevering and judicious training. For all classes of the insane the most important factor in treatment, as well as the most difficult to secure, is a corps of thoroughly competent and conscientious attendants.

“ But few cases of general paresis have been under treatment here—not more than five or six in all—and it will hardly be expected that I should make any contribution to the therapeutics of that not very tractable disease. In epilepsy the bromides are more efficacious than any and all other drugs in diminishing the number of convulsions and the attacks of mental disturbance immediately connected with them, but seem powerless to remedy, and, in most cases, even to check, the progressive mental deterioration so common in this disorder.

" The details of management and discipline in an institution of this kind would have little practical value to any except those engaged in the same specialty. It may not, however, be out of place to state the conclusions to which I have come on some subjects in regard to which there is more or less difference of opinion among alienists.

" Sedatives, such as conium, hyoscyamin, the bromides, and antimony, are scarcely used at all for the purpose of allaying excitement or repressing its manifestations. Any advantage that might be gained in the palliation of symptoms on their continued use seems to me likely to be more than counterbalanced by their deteriorating effects upon the general health. In a few cases an occasional dose of hyoscyamin has had a remarkably quieting effect on noisy and mischievous patients; but it has seemed to me to be more due to their dread of the very disagreeable sensations which the drug produces than to any influence on the morbid process.

" Of hypnotics, chloral, paraldehyde, and sulphonal have been found useful as palliatives in insomnia, and, apart from any benefit to the patients to whom they are given, I believe their use justifiable when necessary to secure needed quiet and rest for others. When it appears desirable to continue the administration of such drugs for any considerable time, it is my practice to change frequently from one to another. Used in this way, in moderate doses, I have not observed bad effects from either of them, although all are doubtless capable of mischief.

" Alcoholic stimulants are used very sparingly. Although I am satisfied that they are of use in some cases, the number is, I think, small, and much mischief may be done by their routine administration.

" In regard to mechanical restraints, the small number of attendants that I am able to employ—not quite one to twenty patients—necessitates its employment, or what, in my opinion, is often more objectionable, seclusion, in a small proportion of cases, not more, on the average, than one per cent. of the number under treatment. I do not, however, believe that, under any circumstances, I should be willing

to dispense with it altogether. Much as its routine use is to be deprecated, what I have seen in visits to institutions which have discarded it entirely has confirmed me in the belief that in some cases it is both more effective and more humane than anything that can be substituted for it."

THE STUDY AND CURATIVE TREATMENT OF INSANITY.

PROPOSED NEW HOSPITAL.

At the weekly meeting of the London County Council at the Guildhall (Lord Rosebery in the chair), Mr. R. Brudenell Carter, chairman of the committee appointed to inquire into, and to report to the Council upon, the advantages which might be expected from the establishment, as a complement to the existing asylum system, of a hospital with a visiting medical staff for the study and curative treatment of insanity, submitted the following report: The committee had received important *vivâ-voce* evidence from sixteen distinguished members of the medical profession, including not only eminent experts in insanity, and physicians chiefly engaged in the treatment of diseases of the nervous system, with which insanity is not necessarily associated, but also physicians and surgeons in more general practice. Printed questions had been sent to every medical superintendent of a public Asylum in England and Wales, as well as to a few other persons of admitted authority, including the medical officer of St. Pancras Workhouse, who has had large opportunities of witnessing the operation of the present system. Sixty-five replies to these questions had been received and analyzed. The committee had arrived at the conclusion that a hospital of the kind described would be likely materially to increase the present knowledge of the nature and causes of insanity, and therefore ultimately to increase the means available for its prevention and for its cure. They were consequently prepared to make the following recommendations:—

"(a) That an adequately equipped hospital, containing one hundred beds, for the study and curative treatment of

insanity in pauper lunatics of both sexes, be established in the metropolis, and that it be under the direction and control of the Council. (b) That the ordinary medical staff of the hospital consist of a chief resident medical officer, who has had asylum experience, of an assistant resident medical officer, of four visiting physicians, each of whom shall hold office as physician or assistant physician in a general hospital, and of a pathologist. The members of this staff to perform such duties, and to receive such stipends or honoraria as the Council may from time to time direct. (c) That, in addition to the ordinary medical staff, the following honoray medical officers be appointed—namely: A surgeon, an ophthalmic surgeon, an aural surgeon, a laryngologist, and a gynæcologist. Each of these honorary officers should hold, or should have held, similar office in a general hospital, and they should all be available as consultants whenever required by the physicians, or for the performance of operations which fall within their respective departments, when such operations are decided upon.” The Committee were of opinion, however, that these recommendations could not be adequately considered by the Council apart from the grounds on which they rest, and also that an account of these grounds should be rendered accessible to the rate-payers, in explanation of the expenditure which would be revuired in order to carry the recommendations into effect. They therefore recommend: “That your committee be instructed to prepare a detailed report upon the question submitted to them, with a sufficient summary of the oral and written evidence which they have received; and that this report and summary be printed prior to their presentation to the Council.”

This report was, after discussion, agreed to.—*The Lancet*.

Dr. H. M. Bannister, of the Illinois Eastern Hospital for the Insane, Kankakee, writes, in reference to the general therapeutical policy of that institution, as follows:

“Only a small proportion of our patients take medicine regularly, not over six or seven per cent. This includes

the sick in the infirmaries, the consumptives, the epileptics, and those taking placebos, as well as the patients who are receiving medicine given for the purpose of directly affecting their mental condition. Even in cases of acute mania any active medication is not always needed or employed; a warm bath and an enema sometimes is sufficient to relieve the symptoms and start them on the road to recovery. Usually, however, in excited cases we give some sedative to procure sleep. Chloral, either alone or in combination with hyoscyamus and conium, or hyoscyamin or hyoscine, hypodermatically, and in some cases the bromides, are the drugs commonly employed. As a rule, we do not give large doses, nor do we continue their use longer than seems absolutely necessary. We have given sulfonal considerable trial, and it has been reported as useful among the female patients; at present it is not much used on the male side of the house. The main ideas in treating acute mania of the active kind are to secure proper rest and nourishment and keep the bodily functions all in order, and all means available for these ends are employed. It is also a usual thing to give a maniacal patient a special attendant and keep him out of doors as much as his condition and the weather will permit.

"In cases of melancholia there is sometimes more steady employment of medicinal treatment, and I have met with many cases in which the continued administration of opium, generally in small tonic doses, was very useful. There is the same or even greater necessity for giving attention to the condition of the bowels in this form of insanity as in mania, and in some cases the mental depression has been greatly relieved, or even done away with, by emptying the colon. I have notes of one or two very striking cases that strongly support Schroeder Vanderkolk's theory of sympathetic insanity from this cause.

"The medical treatment in most of the other forms of insanity is on general principles, but epileptic cases of course receive special treatment, and in cases of general paralysis I have quite generally employed the iodide of potash, and sometimes with great apparent benefit. A very

large proportion of paretics received here have a luetic history, and many of those that lack the history have almost certainly suffered from specific infection.

"Stimulants (alcoholic) are employed only to a comparatively slight extent, and usually only for old and feeble patients.

"As regards other than medicinal treatment, I would say that we employ scarcely any mechanical restraint, and as a rule only mittens that leave the arms free, but prevent the use of the fingers. Probably an average of less than one-twentieth of one per cent. of our patients are under any form of restraint during the year. Seclusion is used to some extent with very violent patients, but the percentage of this is also small.

"Employment, we find, is one of the best specifics for insanity; it helps the patients to forget their morbid fancies and keeps them from bad habits of mind and body. More patients can date their improvement from their first being employed, I think, than from any other one thing. Its benefits are not confined to the curable cases; there is often a great improvement in the chronic and incurable ones. We have been steadily increasing our percentage of employment amongst our patients, and at present it averages about seventy per cent. doing some useful work during at least part of the day.

"Another thing that is very valuable in the management of the insane is open-air exercise, and we keep as many of our patients out of doors, part of the day at least, as is possible in pleasant weather.

"A great deal depends on the attendants in the successful treatment of the insane. Natural fitness is one of the first requisites, but much can be gained by training, and we have endeavored to instruct our attendants so that they may not only care for the patients properly, but also be able to administer special treatment, massage, electrical applications, etc., under the direction of the physicians. We have employed these methods in certain cases in our treatment where they seem to be of use, also special gymnastics, etc.

"You will note that opiates cut little figure in treatment here. While they are used of course as placebos, they are not given as lavishly as in most institutions of this sort. T. S. Clouston, of the Royal Edinburgh Asylum, in his 'Mental Diseases' says: 'Opium I utterly disbelieve in. I performed a series of experiments with it in melancholia, and it caused loss of appetite and loss of weight.' And again: 'In ninety-nine cases in a hundred it lessens the appetite and impairs digestion.' That there is truth in these emphatic and unqualified statements may be admitted, but that they carry with them a true estimate of the value of this *hominibus deorum donum* is not borne out by experience. In melancholia of aged persons especially, it is found curative in not a few cases, to add comfort in most, while it is exceptional for it to lessen the appetite and impair the digestion. In not a few cases the effect is the reverse: it imparts appetite and aids digestion. Wood's experience with the drug agrees much more nearly with observation here.

"Clouston's experience with chloral, as set forth in 'My experience is that it has a subtile influence for harm upon the brain when much given, by which the organ loses that quality which we call tone,' is, it is believed, entirely correct. That the lavish exhibition of it, had recourse to in many institutions as a hypnotic, it is confidently believed is making 'asylum' dementals by the hundreds.

"Recurring to opium, it may be as well to state that it is used here almost entirely subcutaneously and most generally in combination with atropin sulph.; the acetate and bi-meconate is occasionally employed by the mouth. In very small doses— $\frac{1}{48}$, $\frac{1}{32}$, $\frac{1}{24}$, $\frac{1}{16}$ gr.—it is not seldom found a splendid nerve-tonic, especially combined with a corresponding amount of atropin, say $\frac{1}{1000}$ gr. up to $\frac{1}{300}$ gr. ter die.

"In acute mania more reliance is placed on the bromides and cannabis indica, sometimes reënforced by hyoscyamus and conium. In melancholia, *if shut up* to one drug, I should take some form of Clouston's 'utterly disbelieved' in drug, *opium*."

PHYSICAL TRAINING FOR THE INSANE.

(From *American Journal of Insanity*.)

Dr. Walter Channing, of Brookline, Mass., read a paper on this subject before the Association of Medical Superintendents of American Institutions for the Insane.

He has obtained good results from the employment of a long systematic graded course. He employs a professional teacher to conduct the exercises, who is aided by music. He would have the course varied enough to extend over years. The classes were made up from both sexes and attendants. The results have been gratifying directly and collaterally. "There can be no doubt that nutrition has been improved in a number of cases, but the especial perceptible result, which is more marked as time goes on, is the generally improved physical and moral tone among the patients and employés. There is less susceptibility to trifles, more freedom of motion, more independence of action, more appreciation among all of the value of exercise and care of the body, and more coöperation in the general treatment. The amount of amusement furnished is of course considerable. The lively music and brightly lighted hall and magnetic teacher are all diverting, and the exercises are just enough varied to keep up the interest without fatiguing. The results are certainly favorable enough to encourage me to persevere."

In the discussion that followed the reading of Dr. Channing's paper, Miss A. W. Adams, his instructor, gave the following explanation of the method employed: "Our system induces no thought on the part of the pupil, nor real mental effort. The evil we are trying to counteract is the abnormal pressure; consequently pupils work entirely by imitation, which is largely intuitive, and little call is made on the nerve-force. This principle makes our system particularly adaptable to the insane and nervous, as the object is to send through the system the purest blood, with the least possible exhaustion of nerve-force. The work is divided into yearly courses, and is carried on in a slow but progressive manner, commencing with simple elementary work—one movement dependent upon and leading up to

the next, until finally the whole body is exercised without apparent effort on the part of the pupil."

"I have endeavored to carry out these same methods with the insane, with the exception that I am obliged to go much more slowly, often to omit alternate movements, and complicated ones entirely. Instead of giving three or four rests during the hour, as is customary with beginners, I find it is better to keep the class working steadily for thirty or forty minutes, and then dismiss them. By so doing I held their attention better and kept the interest up. With very nervous, delicate patients, who are not strong enough to take the exercises standing, very slow, stretching movements on the back are given, which have a very soothing, quieting effect."

EIGHTEENTH ANNUAL REPORT OF THE STATE HOMŒOPATHIC ASYLUM FOR THE INSANE, AT MIDDLETOWN, NEW YORK.

General statistical table, showing the number of patients treated during the year ending September 30, 1888, together with the results obtained :

	<i>Males.</i>	<i>Females.</i>	<i>Total.</i>
Patients in asylum Sept. 30, 1887, - -	216	239	455
Admitted within the year, - - - -	111	106	217
Whole number of cases treated within the year, - - - - -	327	345	672
Number discharged within the year, -	98	115	213
Number discharged as recovered, - -	40	60	100
Number discharged as improved, - -	13	18	31
Number discharged as unimproved, -	22	24	46
Deaths, - - - - -	23	13	36
Eloped, - - - - -	-	-	0
Not insane, - - - - -	-	-	0
Patients remaining Sept. 30, 1888, - -	229	230	459
Maximum number within the year, - -	-	-	529
Minimum number within the year, - -	-	-	455
Daily average, - - - - -	<u>245³⁵⁴₃₆₅</u>	<u>260⁵⁷₃₆₅</u>	<u>506⁴⁶₃₆₅</u>

Percentage of recoveries on number discharged, - - 46.94
 Percentage of deaths on number treated, - - - - 5.35

DR. SELDEN H. TALCOTT,

Superintendent.

Dr. William A. Macy, formerly assistant physician at the Lunatic Asylum, Blackwell's Island, has been appointed Assistant Superintendent of the Insane Asylum, Ward's Island. Dr. Macy was married to Miss Marion Wright, at New Rochelle, N. Y., on Sept. 3, 1889.

Dr. Allen Fitch, one of the examiners in lunacy for the Department of Public Charities and Correction, New York City, was married to Miss Adelaide Brown, at Williamsport, Pa., Oct. 24, 1889.

Dr. Chas. H. Nichols, superintendent of the Bloomingdale Asylum, has returned from Europe.

Upon the recommendation of Dr. A. E. Macdonald, General Superintendent of the Insane, the Commissioners of Public Charities and Corrections have appointed Dr. E. D. Fisher, Dr. Frank H. Ingram, and Dr. Frederick Peterson, a Board of Pathologist for the New York City Asylums. They have organized with Dr. Ingram, President, and Dr. Fisher, Secretary.

Dr. A. P. Williamson, First Assistant Physician State Homœopathic Asylum for the Insane, Middletown, N. Y., has been appointed Superintendent of the Third Hospital for the Insane, Fergus Falls, Minnesota. This Asylum will be opened sometime during 1890.

Dr. J. E. Bowers has resigned the superintendency of the the Second Minnesota Hospital for the Insane, Rochester, Minnesota, to enter private practice at St. Paul. He is succeeded by Dr. Arthur F. Kilbourae, formerly Assistant Physician First Minnesota Hospital for the Insane, St. Peter, Minnesota, and previously the N. Y. City Hospital on Ward's Island.

The following changes also have taken place in the staff: Dr. Homer Collins and Harry Randall, Assistant Physicians, have resigned, to go into private practice; and Drs. Nathan Baker and Sarah Linton have been appointed in their places. The hospital is about to open a large "annex" for women, to correspond with one it already has for men. It will hold 175.

Dr. P. M. Wise has resigned the superintendency of the Willard Asylum, N. Y., to become superintendent of the St. Lawrence State Asylum, at Ogdensburg, N. Y.

Dr. D. D. Richardson has been appointed superintendent of the New State Hospital for the Insane, Farnhurst, Delaware.

Dr. Eugene Grissom has resigned the superintendency of the North Carolina Insane Asylum. Dr. William R. Wood was appointed to fill this vacancy. Dr. G. R. Pearsall was appointed Assistant Physician.

Dr. C. E. Wright has been appointed Superintendent of the Indiana State Hospital, at Indianapolis, in place of Dr. T. S. Galbraith resigned.

Dr. G. G. Shanks resigned the superintendency of the Kings Co. Asylum. He has been succeeded by Dr. Walter S. Fleming, formerly Assistant Physician at Branch Asylum, St. Johnland. Dr. Fleming's marriage to Miss Lilian Van Dolsen, of New York City, is announced for December 3, 1889.

**Which is the Most Powerful ?
and the Most Reliable ?
of All Pepsins ■**

Merck's Pepsin 1:2000

— SCALE OR POWDER —

SEE "MERCK'S INDEX," PAGES 106 AND 167

THE
Journal
OF
Nervous and Mental Disease.

THE STEVENS COMMISSION.

HISTORY OF THE INQUIRY THAT LED TO THE
APPOINTMENT OF THE COMMISSION.

In the early part of February, 1887, Dr. C. L. Dana, then president of the New York Neurological Society, sent to Dr. Stevens the following letter :

50 West 46th Street, N. Y.,
February 6th, 1887.

Dear Doctor :

I wish very much that you would consent to read a paper at the next meeting of the Neurological Society, March 1st, on the relation of ocular disturbances to nervous disease, or some such title.

We should like very much to have you embody the substance of your Belgian Prize Essay.

Can you not do it? I can assure you of an interested audience. I spoke to Dr. Ranney about the matter ; but he thinks you ought to speak first.

I have been trying to call upon you personally and discuss the matter, but I trust this note may be sufficient.

There is a bare possibility of the March meeting being engaged, but in that case you could have the meeting in April.

I express the unanimous feeling of our Council in hoping you will accept.

Very truly yours,

C. L. DANA,

Pres. N. Y. Neurological Society.

Dr. Stevens wrote to Dr. Dana accepting this invitation, and his acceptance was thus acknowledged :

50 West 46th Street,
February 13th, 1887.

My Dear Doctor :

I am very glad to find you will be able to read the paper. Will you kindly send me the exact title soon.

Very sincerely,
C. L. DANA.

Send me also the names of such gentlemen as you would like to have discuss it.

The title of Dr. Stevens's paper which was read to the Society March 1st, 1887, was :

"Irritations arising from the visual apparatus considered as elements in the Genesis of Neuroses."

The central thought contained in this paper was embodied in a proposition already included in a memoir which had been submitted to the Royal Academy of Medicine of Belgium, which memoir has been published by that distinguished body. The proposition was as follows :

"Difficulties attending the functions of accommodating and adjusting the eyes in the act of vision, or irritations arising from the nerves involved in these processes, are among the most prolific sources of nervous disturbances, and more frequently than other conditions, constitute a neuropathic tendency."

The doctrine thus announced was advocated in a clear and impressive argument, and the hypothesis was verified by cases illustrative of the results of practice. There was in the paper no claim that ocular defects are the sole cause of neuroses, but the view that such defects constitute an important element in the pathogeny of functional nervous troubles was strongly presented.

In the course of the discussion which followed one member of the Society moved that a Committee of the Society be appointed to examine all the cases which had been presented as illustrative examples, with the view of ascertain-

ing the truth of the statements. The motion was not carried, but at a later time the same member renewed the subject.

Dr. Stevens suggested that any inquiry for the purpose of determining the value of his proposition should start with cases studied before as well as during and after treatment. He asserts that he did not express at that time a willingness to enter upon such an inquiry, but he soon after that received the following letter:

50 West 46th St., New York City.
March 16th, 1887.

Dear Doctor:

Dr. Seguin said that you would be willing to have a committee of the Neurological Society examine cases submitted to your treatment. Accordingly, at a meeting of the Judicial Council held at my house March 16th, a committee of five was appointed to confer with you. The gentlemen on the committee are Drs. Seguin, Birdsall, Starr, W. O. Moore and David Webster.

All of these gentlemen are anxious to learn the truth regarding the efficacy of your methods, and I think that a report valuable to science and creditable to yourself will result.

Will you kindly inform me if you will co-operate?

Very truly yours,

C. L. DANA.

From this letter the following correspondence resulted.
C. L. DANA, M.D.,

My Dear Doctor:—Your favor dated March 16th was received to-day, and I hasten to reply that I shall be glad to co-operate with the Neurological Society in an inquiry in regard to the value of the doctrine advocated in my paper, read to the Neurological Society, March 1st, to be conducted in such manner as to be consistent with justice to all immediately concerned in the inquiry, and satisfactory to the medical profession.

To make such an inquiry of true value, certain conditions should be observed, and those which suggest themselves to me I will state here. They are substantially those suggested to Dr. Seguin in our conversation at the close of the meeting, March 1st, but rather more in detail.

1st. To make such an inquiry of substantial value, the results of treatment in a number of cases of important neurosis should be observed from the beginning; the precise character of the neurosis should be fully established and the difficulties which may have already attended well directed treatment should be fully understood.

2d. The Commission should examine and select such a number of cases as may be determined upon (perhaps not less than twelve nor more than twenty) which should be typical cases of some of the most important functional nervous diseases and which should have during many years resisted approved methods of treatment (unless the Commission should in special cases decide that a less time would represent a reasonable duration of the complaint for such an inquiry). The inquiry should extend to such classes of diseases as epilepsy, insanity, chronic chorea, or other representative neuroses, not complicated with known organic disease or conditions which would modify the prognosis or treatment.

3d. The cases selected should be decent in manner and dress, and should be sufficiently tractable, and should possess sufficient intelligence to enable one in charge to form correct judgment concerning the ocular conditions.

4th. The treatment of the cases should be conducted by methods directed to the relief of ocular defects and difficulties; all medicines and other forms of treatment to be withdrawn, except that such medical treatment as might be demanded for intercurrent temporary conditions should be afforded upon conditions to be prescribed by the Commission. If no important ocular defects should be found, the fact should be reported to the Commission, when the case would be considered as concluded. The times and place of treating the cases should be so arranged as to occasion least inconvenience to one upon whose time and strength the demands are already excessive.

5th. The Commission should, before accepting any case as one to be included in the inquiry, carefully examine it and have a complete record made of the past history and of the existing physical or mental condition, not only in respect to the most important affection, but in respect to collateral states which any of those engaged in the inquiry should deem of importance. The record should also include a history of preceding treatment so far as it may be known, a copy of the record to be kept by the Commission and one by myself.

6th. Photographs of every patient should be obtained in every case in advance and at such subsequent time as

may be agreed upon, such photographs to be taken under circumstances which would fairly represent the physiognomy of the persons at the time of taking; the negatives should not be "retouched."

7th. At a time to be agreed upon in advance, the Commission should report to the Neurological Society the condition of the several patients with a statement of the comparative physical or mental state of each in all respects, with that at the beginning of treatment. They should make a general statement of results up to that time. A second report of similar character should be made some (perhaps six) months later. Such reports should be for publication, and copies in advance of submission to the Society should be furnished to me. Their report should also be made to the Commission of the methods and detail of treatment of each case, together with its progress and the conditions remaining at the time of making the report.

8th. Should any patient decline or neglect treatment or withdraw without the consent either of a majority of the Commission or of myself, such case should not be regarded as included in the inquiry.

9th. The Commission for such an inquiry should consist of gentlemen not only well known to the medical profession, but who have not publicly assumed an attitude so adverse to my views or to myself personally, that their judgment might be influenced by such a circumstance. At least two members of such Commission should be suggested by myself.

Believing that an inquiry conducted upon principles strictly just and thoroughly scientific would be of interest and of value to the profession, which we would all gladly serve, I shall be glad to co-operate with the Neurological Society upon conditions such as are outlined above.

Very respectfully yours,

GEO. T. STEVENS.

33 West 33d St., New York,
March 18, 1887.

50 West 46th St., New York, March 21.

MY DEAR DOCTOR:

Your conditions are perfectly just and fair in my opinion, except that it is impossible that you should have any share in naming the members. If, however, any of them are in your opinion too openly biassed against you, please let me know, and I will try to make some change.

Unless I hear from you, however, the Committee remains Drs. Seguin, Birdsall, Webster, Moore and Starr.¹ I can assure you that all are anxious to be entirely just to you.

Very sincerely,

C. L. DANA.

33 West 33d St., New York.

C. L. DANA, M. D.,

My dear Doctor :—It is but just to say that two of those named for your Committee have, by published statements, done me much injustice. I am, however, greatly desirous of promoting the inquiry, and I freely withhold my objections to them and will gladly coöperate with them. If so much is conceded by me, will not a generous profession consent that, in an inquiry which must be so important to myself at least, one or two men, positively known not to be biased adversely to the views to be subjected to inquiry, should act with such a Committee, which might perhaps be enlarged to that extent?

This inquiry has been asked for not by myself, but by the Neurological Society. In consenting to it, as I gladly do, I assume a great deal of extra care and labor in the hope that I may be performing a duty and a service to my profession. Is it necessary, under the circumstances, that there should be excluded from participation in the inquiry any person who has shown a willingness to judge kindly of the subject under consideration?

Such additional members need not of necessity be personal friends or even personal acquaintances of myself, but I believe that I should have reason to be satisfied with them in the above respect.

Very sincerely yours,

GEO. T. STEVENS.

March 23, 1887.

50 W. 46th St., March 25, 1887.

Dear Doctor :—Please name the two gentlemen whom you wish added to the Committee, and I will add one or both if I can do so. They must be members of the Neurological Society, of course.

I feel sure that the inquiry cannot fail to redound to your credit and that it will be of great advantage to the

¹ Dr. Frank P. Foster and Dr. Dana were subsequently added to the Committee.—ED.

profession. I quite appreciate the spirit you are showing in the matter.

Very truly yours,

C. L. DANA.

The Commission being constituted, meetings between members representing it and Dr. Stevens were arranged with the view of establishing a basis for the inquiry. The plan which had been presented by Dr. Stevens did not meet the views of some of the members, and demands were made for concessions to the wishes of these. Among these demands were several to which Dr. Stevens felt that he could not concede. Among them was a proposition that no case should be sent to him until examination of the ocular conditions had been made by the Commission, when the Commission should be at liberty to send the patient or not. A considerable correspondence grew out of these propositions, of which a single letter from Dr. Stevens (reproduced here) is sufficient to show something of the nature :

33 W. 33d St., N. Y., April 4, 1887.

Dear Dr. Starr:—On reading your letter of to-day, I find more reason than ever for insisting upon what I have written in respect to ocular examinations preceding my own.

It did not occur to me as possible that the Commission should think of culling cases in respect to the existence or non-existence of ocular conditions before submitting them to me. My conditions are explicit in stating that if no important ocular defects are found, of course by myself, "the fact should be reported to the Commission, when the case would be considered as concluded." Such a case would most certainly be counted and against me. To allow a Commission to select in advance, on the ground of the existence or non-existence of ocular defects, would be a most unusual proceeding. I most emphatically protest against any case being accepted or rejected on the ground of the presence or absence of ocular defects; and I also renew my objection to any interference with the accommodation of the eyes or familiarizing the subjects of the inquiry with the phenomena attending ocular muscular tests.

I am, Doctor, very sincerely yours,

GEO. T. STEVENS.

At length, about the first of May, a plan was finally adopted, and the work of the inquiry commenced.

Dr. Stevens claims that the understanding of the articles of the agreement was, that the number of cases agreed upon as the basis of the trial were to be sent to him within a very short time, and that a preliminary report, showing the condition of all the persons subjected to the inquiry, should be made early in November, 1887. His understanding was that this should in fact mark the formal beginning of the investigation. Dr. Stevens claims that the minimum number of cases had not been sent to him at the end of the first year, nor even at a much later time. This question is presented on one side by the Commission in its report, and on the other by Dr. Stevens in his reply.

A copy of the Commission's Report was forwarded to Dr. Stevens with the following letter :—[ED.]

October 28th, 1889.

DR. GEO. T. STEVENS,

Dear Sir :—At a meeting of the Commission, held Oct. 27th, your letters of the 23d and 25th inst. were carefully considered and the statements discussed. It was unani- mously concluded that the Report should be presented to the Society on November 5th, 1889. A copy of the Report as adopted by the Commission is herewith sent to you. Addenda relating to the present condition of the eyes of the cases accessible to the members of the Commission, and in preparation by the ophthalmologists, and will be read before the Society. These will be furnished to you, if you so desire, when received by the Secretary.

Yours truly, M. A. STARR.

REPORT OF THE COMMISSION

ON THE

TREATMENT OF EPILEPSY AND CHOREA BY
THE CORRECTION OF OCULAR DEFECTS

MADE TO THE

NEW YORK NEUROLOGICAL SOCIETY,

*November 5th, 1889.*¹

MR. PRESIDENT :

The Committee known as the "Stevens Commission," appointed two and one-half years ago, beg leave to submit the following report to the Neurological Society.

INTRODUCTORY REMARKS.

At a meeting of the Society held March 1, 1887, Dr. Geo. T. Stevens, of this city, read a paper entitled, "Irritations arising from the Visual Apparatus considered as Elements in the Genesis of Neurôses."² Great interest was manifested in the hearing of this essay, as it was a *résumé* of the author's work during many years; a work characterized by

¹ The first copy of the reports obtainable by us was the one sent officially to Dr. Geo. T. Stevens by the Commission. It was not till this copy had been set in type that we were requested to compare and arrange this report in conformity with the one in possession of the Commission. The ophthalmological addenda spoken of in Dr. Starr's letter to Dr. Stevens have been incorporated in the report.

Differences in the report are carefully noted in italics, which are those of the Commission, and any corrections that were made in the report since read (November 5th) have been separated by the aid of the Secretary of the Commission and placed in foot-notes, also in italics. We have compared and adjusted slight typographical differences with the aid of the Secretary of the Commission.

Exhibits will not be published. The full histories will appear in the December number.—EDITORS.

² Published in the New York Medical Journal, April 16, 1887.

exactness of method, great perseverance, and remarkable satisfaction with the results obtained. While the author disclaimed the pretension that ocular defects and consequent nervous strain were the sole cause of the great neuroses, chorea, epilepsy, and hystero-epilepsy, he yet advanced the proposition that these defects and the consequent strain were such potent exciting causes of these diseases, that their correction would in many cases lead to cure, or at least relief. The interest of the discussion which followed the reading of the paper lay in the relation of eye-strain to the genesis of chorea and epilepsy. The author's views were so novel and extreme, his allegations of good results so strong, that there was a general expression of a desire to thoroughly test his methods. Simple negation, based on different personal experience with these two diseases seemed of little value, and a number of members of the Society suggested that a fresh series of cases of chorea and epilepsy be treated by Dr. Stevens, under the supervision of a committee, and that a report of results be made in due time. It is hardly necessary to state that every member of the Society was disposed to welcome a new treatment of the neuroses, if its efficacy could be established upon concurrent testimony. This feeling led to the appointment of the Commission to co-operate with Dr. Stevens in the test of his method upon new cases.

STATEMENT OF DR. STEVENS' ALREADY PUBLISHED VIEWS.

It may be well to state here, very briefly, Dr. Stevens' views as regards the etiology and therapeutics of chorea and epilepsy. We quote from the paper read before the Society.

"Difficulties attending the functions of accommodation and of adjusting the eyes in the act of vision, or irritations arising from the nerves involved in those processes are among the most prolific sources of nervous disturbances, and, more frequently than other conditions, constitute a neuropathic tendency." This proposition which had been submitted (supported by an elaborate essay) to the Royal Academy of Medicine of Belgium in 1883, presumably well

illustrates Dr. Stevens' views as to the etiology and pathogeny of neuroses. Several cases are related in these two publications showing good results, cures and ameliorations, in a number of cases of chorea and epilepsy.³

As it is of great importance to have a more definite idea of Dr. Stevens' results in chorea and epilepsy, we must quote from a book published by him in 1887, entitled "Functional Nervous Diseases, their Causes and Treatment," which, we assume, sums up the author's whole experience to date, *i. e.*, up to the time when the Commission began its conjoint study with Dr. Stevens. With respect to *chorea*, the following brief *résumé* may be made. As regards the relation of ocular defects to the disease, Dr. Stevens states that he has examined 118 cases, and found defects as follows:

Simple hypermetropia,	-	-	-	-	in 78 cases.
Hypermetropic astigmatism,	-	-	-	-	in 13 "
Mixed astigmatism,	-	-	-	-	in 5 "
Myopia, unequal in two eyes,	-	-	-	-	in 6 "
Myopic astigmatism,	-	-	-	-	in 11 "
Insufficiency of the lateral recti muscles,					in 5 "

It is also stated that in "a considerable number" of the cases with refractive errors, there was more or less muscular disability. Thus it appears that not one of 118 subjects of chorea, according to Dr. Stevens, presented a normal ocular apparatus. Dr. Stevens concludes:⁴ "After a careful study of the cases which have been examined, and treatment of many of them for the removal of ocular anomalies, the author does not hesitate to assert the direct relation

³ This publication in book-form includes the substance of the Brussels Essay, which has since been published by the Royal Academy of Medicine of Belgium, among its "Memoires des Concours et des Savants Etrangers, Vol. VIII., fasc. 3, 1888;" under the author's title of, "Essai sur les Maladies des Centres Nerveux, leurs causes et leur traitement; Irritation oculo-neurale." The essay read before the Neurological Society is really of later date and contains additional private cases and others observed at the Willard Asylum for the Insane, New York.

⁴ Op. cit., p. 93. ⁵ Op. cit., p. 92. ⁶ Op. cit., p. 93. ⁷ Op. cit., p. 100.

⁸ Functional Nervous Diseases, p. 105.

between these ocular difficulties and the disease in question." With respect to therapeutic results, we note as follows. Dr. Stevens says:⁵ "It can be easily understood, then (after having shown the frequency of hypermetropia), why chorea, in the ordinary cases, ceases, as does asthenopia, but somewhat less promptly, upon the discontinuance of the use of the eyes for close work." Hence the apparent advantage of withdrawal from school. The remarkable assertion is also made that "a supposed cure of chorea" (by ordinary means in several of his cases) "has only been a change to chronic headache or other neuroses."⁶ Eight cases, all successful, are reported in the three essays, several appearing in two of the essays. Two of these eight cases were only improved. No statistics are given, but the statement is made that many other cases have been cured. Upon these data the author says:⁷ "May we not conclude that chorea is emphatically a nervous trouble depending upon ocular conditions?"

With respect to *epilepsy* we are informed that the following ocular conditions were found in 100 consecutive cases:⁸

Hypermetropia (including hyp. astig.),	-	in 59 cases.
Myopia (including myopic astig.),	-	in 23 "
Emmetropia (or errors less than 1 D.),	-	in 18 "

"In the greatest number of cases examined in private practice very marked insufficiency of the motor muscles of the eyes was found, and it may be here observed that, so far as ocular irritations are concerned in the origin of a tendency to epilepsy, muscular irritations are doubtless much more efficient than refractive anomalies."⁹

As regards therapeutic results we would simply present the following quotation from the paper read before the Society: "Of sixty-four consecutive cases of well marked epilepsy in private practice, of which in every instance the disease had been of more than one and in most of many years' duration, and in all of which the treatment has been directed to ocular conditions, medicines having been,

⁹ N. Y. Medical Journal, *l. c.*—p. 426.

except in a single instance discontinued, thirty-two have remained free from attacks for a time varying from several months to several years (a foot-note states that 'several of these patients have remained well for a period of from five to seven years'), a time which would in all ordinary conditions enable us to regard the cases cured. Twenty-one have shown under this treatment such marked improvement as to indicate with certainty that the ocular conditions and the disease were in relation as cause and effect. In some of these cases the change has been very remarkable, but short of absolute relief. In eleven cases no improvement has occurred, or, if any, of only temporary character. Thus, without the employment of drugs to destroy the nervous susceptibility to irritating causes, 50 per cent. of these patients are, so far as can be known, well; another large proportion much better off than while using bromides, while only 17 per cent. show no improvement." In this three essays upon the subject Dr. Stevens has given brief summaries of the history of only 17 cases, 13 from his private practice, and 4 from the Willard Asylum for the Insane. Of these, ten cases are reported as "cured."

It will thus be seen that as regards chorea Dr. Stevens leads us to believe that he considers it essentially dependent upon ocular conditions, but gives no numerical statistics showing the proportion of cures; and that as regards epilepsy he reports about 50 per cent. of cures through treatment of the ocular conditions.

PLAN OF THE INVESTIGATION.

The first proposition unofficially made to Dr. Stevens after the reading of his paper, by a member of your Commission, was that his recorded cases should be sifted and reported upon to the Society. This suggestion, not altogether unreasonable in view of the extraordinary results claimed was instantly rejected, as implying a doubt of Dr. Stevens' veracity. Then it was proposed that new cases of chorea and epilepsy should be furnished to Dr. Stevens by members of the Society and their treatment watched, recorded and reported upon by a committee of the Society,

acting conjointly with the author of the paper. This was accepted and your Commission duly appointed. It organized March 26, 1887, with Dr. E. C. Seguin as Chairman and Dr. M. Allen Starr as Secretary. After repeated conferences with Dr. Stevens, and considerable debate as to modes of procedure and mutual concessions, the following plan of agreement was finally adopted. It is as follows :

PLAN OF PROCEEDINGS IN THE INVESTIGATION REGARDING DR. STEVENS' METHODS OF TREATMENT IN FUNCTIONAL NERVOUS DISEASES, ADOPTED BY THE NEUROLOGICAL SOCIETY'S COMMISSION.¹⁰

1. The subjects to be investigated shall be functional nervous diseases, of which epilepsy and chronic chorea are especially selected, the Commission being satisfied that these are typical neuroses, and admitting that reasonable success in their treatment will establish the proposition that neuroses are curable by the correction of ocular defects. The number of cases subjected to treatment shall not be less than twelve or more than twenty.

2. Typical cases only of these diseases shall be chosen, which are not complicated by any known organic disease, and which have resisted approved methods of treatment for a reasonable time. The precise character of the neuroses shall be fully established by independent examination by two members of the Commission, and disputed cases shall be excluded from treatment. The patients shall be sufficiently intelligent to enable correct judgments regarding the ocular conditions to be reached. They should be sufficiently neat in their appearance to be received in a private office.

3. General medical histories of each case shall be recorded, and a copy filed with the Secretary open to inspection by all members of the Commission. A copy of the history shall be furnished to Dr. Stevens with the case. Such histories shall include a complete medical record of the case, of the existing physical and mental condition, not only in respect of the most important affection, but also in respect of collateral states which any of those engaged in the inquiry shall deem of importance. The record shall also include a history of preceding treatment so far as it may be known. Photographs may be obtained of any or all patients, at Dr. Stevens' option, before and after the ocular treatment, the negatives not to be "retouched" in either case.

¹⁰Incorporated in the report by consent of the Secretary of Commission.

4. The first ophthalmological examination of any cases shall be made by Dr. Stevens in the presence of at least one member of the Commission. The cases submitted to Dr. Stevens are not to be selected because of the presence or absence of known ocular defects. Subsequent to Dr. Stevens' first examination, and prior to operation, the ocular condition may be investigated by any member of the Commission. When Dr. Stevens has by one or more examinations determined the nature of the ocular defect for which an operation is necessary, he shall record the same prior to the operation and deliver the record to some member of the Commission. The result of the examination by the ophthalmological members of the Commission shall also be recorded; and these records shall be filed with the Secretary. At least one member of the Commission may be present at all operations, due notice (forty-eight hours) of such operation having been given by Dr. Stevens to the Secretary. But the absence of members is not to prevent Dr. Stevens from proceeding with the operation, provided a reasonable time be given (fifteen minutes) for the appearance of the members notified. Notifications of operation are to be sent to the ophthalmological members of the Commission and to the member whose case is to be operated upon, by the Secretary.

5. A report shall be made by Dr. Stevens to the Commission of the conditions found of the methods and details of treatment, and of the progress of the case and the conditions remaining at the time of making the report. If any patient declines or neglects treatment, such case shall not be regarded as included in the inquiry or counted. All cases examined by Dr. Stevens shall be classified in two categories: first, an examination shall be made to show how many of the cases sent presented ocular defects; second, an examination shall be made of the cases with ocular defects to show the results of treatment. The two categories may be separately or comparatively utilized. All medicinal treatment shall be suspended while the patient is under treatment by Dr. Stevens, unless intercurrent conditions arise requiring attention, and such shall be noted in the history.

6. The Commission shall report to the Neurological Society at its meeting in November, 1887, and subsequently as it may decide, the comparative physical and mental state of the patient before and after treatment being stated in such reports. A copy of each report shall be furnished to Dr. Stevens prior to its submission to the Society.

7. The Commission shall consist of three neurologists, and two ophthalmologists, appointed by the President of the Neurological Society and of Drs. E. L. Dana and F. P. Foster, selected by Dr. Stevens.

8. The times and places of examining and operating upon the cases shall be or arranged as to occasion no inconvenience to Dr. Stevens. The members of the Commission agree to be present at the times and places appointed so far as it is in their power.

DIFFICULTIES ENCOUNTERED BY THE COMMISSION.

The clear intent and meaning of par. 4 and 5 are, we believe, that Dr. Stevens should furnish copies of his notes of cases to the Commission for filing, either through "some member of the Commission," or directly through the Secretary.

This interpretation of the clause has on several occasions been brought to Dr. Stevens' attention by the Commission, but he has persistently declined to furnish the data in question.

At a meeting of the Commission held Oct. 7, 1888, it was resolved that "Dr. Stevens be requested to forward notes of the cases under his care to the Secretary." A correspondence ensued between the Chairman and Dr. Stevens (*vide* Exhibit A), at the close of which Dr. Stevens positively refused to furnish the data requested. Dr. Stevens' last note on the subject, dated Nov. 29, 1888, closes as follows: "I may add that there is nothing in the agreement which calls for a report from me until the time for a final summing of the work arrives. Permit me also to remind you that you have had, from time to time, reports of the ocular conditions of these patients. The conditions at the beginning were observed and recorded both by the oculists and myself. Our records agree, and they report the result to the Secretary. At each operation, if one is made, the oculists again make a record of the conditions before and after the operation, and this record also agrees with mine. Beyond this I must absolutely decline to send to the Commission running reports of the work. The book of these cases is always open to the oculists."

Signed,

GEO. T. STEVENS.

If any one will compare this statement with the provisions of Par. IV. and V. of the Agreement, he will be at once convinced that Dr. Stevens violated both the spirit and the text of the compact. Inasmuch as Dr. Stevens has not furnished the Commission with reports of the cases, or copies of his various notes of them (or allowed of copies being made at the Commission's expense, as was proposed), it will be evident the Commission has labored under serious difficulties.

In addition to refusing notes of the cases, Dr. Stevens has retained in his possession the only existing notes of one case, to the serious inconvenience of the Commission. (*vide* Exhibit B.)

In Dr. Stevens' final reply to the Commission's request for copies of all notes of cases for filing in the Secretary's hands, it is stated that "the book of these cases is always open to the oculists."

Acting upon this invitation, the Commission in December, 1888, delegated Dr. Wm. Oliver Moore to go to Dr. Stevens' office and copy or cause to be copied at the Commission's expense the records of the cases treated by Dr. Stevens. This Dr. Moore was not permitted to do.

As provided for in the agreement your Commission made a report in November, 1887, but this was of necessity only a report of progress. During the autumn of 1888, as numerous cases had been under treatment by Dr. Stevens for many months, it was discussed at several meetings of the Commission when a final report should be made. Preliminary to the planning of a report it was necessary to collect all the data relative to cases which had been sent to Dr. Stevens. This was made difficult by Dr. Stevens' refusal (on November 29th) to furnish his notes of the cases. Prior to this an attempt was made by your Commission to induce Dr. Stevens to fix a limit of time of treatment as determining the success or failure of treatment in any given case. At a meeting held April 8th, 1888, it was resolved: "That the Secretary obtain a statement from Dr. Stevens as to the minimum and maximum limit of time which he will consider satisfactory for cases to be submitted to him

in future by the Commission." For certain reasons the Chairman was ultimately delegated to see Dr. Stevens about this matter. The following is Dr. Seguin's report to the Commission of this consultation, dated April 14, 1888 :

"To the second request of the Commission, that he should state a minimum and maximum time of treatment for purposes of report, or even of record in the Commission's minutes, Dr. Stevens returns a decided negative. After further conversation, Dr. Stevens proposed that when twelve cases shall have been placed under treatment, all together, a report should be made when the last case admitted to the list shall have had six months treatment." (Exhibit C.)

It was evident to the Commission that under the last-named condition a report would be indefinitely postponed, or indeed never made, inasmuch as patients were constantly dropping out.

In considering the request of the Commission for an agreement as to limit of trial of treatment, reference should be made to Dr. Stevens' publications. In these, the reported cases improved immediately after the operation, or after the correction of errors of refraction, and the statement is made that within a few months after treatment no convulsions recurred, and no choreic movements remained ; but this is not supported by definite chronological data.

Dr. Stevens has found fault with the Commission because a large number of cases was not furnished him for the experiment. The members of the Commission exerted themselves honestly to send as many cases as possible to Dr. Stevens ; and in April, 1888, the Commission sent printed postal cards to all the members of the Neurological Society, requesting them to send in cases for the advancement of the inquiry. This appeal proved almost useless. Furthermore, from the first the Commission has accepted such cases as Dr. Stevens himself selected, after examination by members of the Commission in accordance with the agreement, to the number of six. In fact some members of the Commission were at times obliged to use much per-

suasion, and, in many cases, to argue in favor of the trial in order to induce their patients to go to Dr. Stevens. More than that, in several instances, when patients sent to Dr. Stevens grew discouraged by the failure of the treatment, members of the Commission used all their powers of persuasion to cause them to persevere. We can truly say that in this respect we have earnestly tried to favor the efficiency of the inquiry. Lastly, the members of the Commission have refrained, sometimes under very trying circumstances, from publicly expressing any opinions as to the progress of the investigation. If Dr. Stevens has not had as many cases sent to him as he had expected, it has certainly not been through any lack of coöperation on the part of the members of the Commission.

REASONS FOR REPORTING AT THE PRESENT TIME.

The Commission regret to be obliged to report without Dr. Stevens' co-operation, but it seems to us that further delay would be discourteous to the Society, which has waited so patiently, and unfair to the medical profession, which, we have reasons to believe, is anxious to know how Dr. Stevens' method has resulted, and whether it can be recommended for the treatment of epilepsy and chorea.

In the month of April last it was proposed to prepare a report on these grounds, but some members of the Commission thought that nothing would be lost if we granted a request made by Dr. Stevens, through Dr. Birdsall, that more cases be furnished, and the report postponed until November.¹¹ This was agreed to.

This limit of time has now expired. Four new cases have been sent to Dr. Stevens, and three of these have remained under treatment up to the present. Dr. Stevens himself added no cases to this series.

It will be observed that Dr. Stevens' letter¹² of last April

¹¹ COPY OF DR. BIRDSALL'S LETTER TO THE SECRETARY OF THE STEVENS COMMISSION:—"Dr. Stevens stated that if we would send him four acceptable cases within a month, he would agree to a final report in November, provided one or more of the four patients continue until November, but not if they drop out. Sincerely yours," W. R. Birdsall. April 23, 1889.

¹² The letter referred to was Dr. Birdsall's letter, not Dr. Stevens' letter.

(*vide* Exhibit D), contains two distinct propositions. One states that he would like four more acceptable cases; the other that: "one or more of the four patients continue under treatment until November." Of these two conditions the important or vital one is the latter. It would certainly make no difference how many acceptable cases were sent to Dr. Stevens, provided "one or more remained under treatment." This has taken place. Three (3) of the cases have so remained under treatment, and this fact, together with the general reasons already stated, led the Commission at a meeting held Oct. 13th, to decide to submit this report.

Still, we desired Dr. Stevens' co-operation, and to this end Dr. F. P. Foster was delegated at the meeting of Oct. 13th to see Dr. Stevens and ask whether he would now be willing to furnish the Commission with the data in his possession. Dr. Foster reported as follows (Exhibit E): "Dr. Stevens still declines to furnish us with the notes of the progress of the cases. He does not demur to the dates on the list, but he made remarks about two of the patients that I have added in red. He made out a list himself (which I herewith inclose) of those patients that he is willing to have counted." (Signed), FRANK P. FOSTER.

Dr. Stevens upon learning that the Commission had decided to present a report, sent to the Secretary, under date of Oct. 23d, a written request that more time be allowed for the further observation and treatment of the cases, and that the report be postponed until March or April of next year (Exhibit G). This proposition was carefully considered at a special meeting held Oct. 27th, and by a unanimous vote it was decided to report at once.

The data in possession of the Commission are imperfect and in some respects unsatisfactory. This is not the fault of the Commission, but is solely owing to Dr. Stevens' withholding of his notes.

Altogether, 28 cases have been placed on the list of accepted cases (six of these being furnished by Dr. Stevens). The following summary of these cases has been prepared by Dr. Starr, and will serve as the basis for the conclusions of the Commission.

THE WORK OF THE COMMISSION.

After the plan of proceedings had been agreed upon the members of the Commission began to send patients to Dr. Stevens in April, 1887.

Each patient was examined by two neurologists, who agreed upon the diagnosis and compiled a history of the case. The patient was then sent with the history to Dr. Stevens, who examined the ocular conditions. Subsequently the ophthalmologists examined the eyes and reported their results to the Secretary, who added these notes to the copy of the history in his possession. When an operation was undertaken notice of it was sent by Dr. Stevens to the Secretary, and he in turn notified the ophthalmological members and the neurologist who had sent the patient. These members then attended the operation and noticed the conditions found in the eyes before and after it, sending their records to the Secretary. Their non-attendance, however, did not prevent operations from being done, and it is the records of these operations as well as records regarding prisms and glasses prescribed by Dr. Stevens for the patients, which Commission has been unable to obtain.

The patients have from time to time visited the members of the Commission by whom they were sent to Dr. Stevens, and notes of the condition found have been made and sent to the Secretary. In the cases of epilepsy the record of attacks has not in all cases been kept in duplicate, Dr. Stevens having the record on the blanks furnished by the Commission, and in these cases the records have not been accessible.

The following brief summary of the history of each case is given in order that the Society may be informed of the character of the cases selected and of the result in each case.

The cases are arranged in two classes :

First.—Those which are counted by the Commission in accordance with the agreement.

Second.—Those which have been withdrawn and are not counted.

CASE 1.—Elizabeth C., æt. 13½; disease, chronic chorea; Dr. Seguin. This girl, who had had one attack of chorea had not recovered from her second attack, which had become chronic, and had been continuous from September, 1885, to April, 1887, when she was sent to Dr. Stevens. There was found an ocular insufficiency which was treated by the use of prisms and by operations. The first improvement appeared 30 weeks after beginning treatment. Since that time very little continuous improvement has occurred. At times the chorea has been worse, at times she has been almost free from chorea. For the past two months, September and October, 1889, she has been free from chorea, except slight manifestations in tongue and hands for a few days before each menstrual period. She is not cured, as there are occasional slight recurrences of chorea. She is greatly improved. But such periods of improvement having occurred under other forms of treatment. Dr. Seguin expresses his deliberate opinion that the ophthalmic treatment has had very little if anything to do with the improvement. She has had 13 operations, the last being done March 22, 1889, and has worn 8 different forms of glasses. At present she has diplopia for near and far distance, an ocular defect acquired during treatment.

OPHTHALMOLOGICAL REPORT.

“ R. V. = $\frac{20}{15}$; Hm. o. 25 D.

L. V. = $\frac{20}{15}$; Hm. o. 25 D.

Hyperphoria 0°, exophoria 1°, in accommodation 4°, sursumduction R. $\frac{3}{4}$ °, L. $\frac{3}{4}$ °, abduction 5°, adduction 16°. Opthal. normal; movements of eyes do not seem limited in any direction; no diplopia on testing with red glass. Patient says she has had thirteen operations; that when she looks up suddenly from reading she sometimes sees double, and that her eyes smart and burn and run water she reads. Her mother says she does not complain of it very often. There have been very few manifestations since last May.”

Oct. 29, '89.

D. WEBSTER.

CASE 2.—Flora K., æt. 20; disease, epilepsy; Dr. Starr. This patient, who has well marked epilepsy, had been under

observation for four years during which time under treatment she averaged about five attacks a month of grand mal of varying degrees of severity—only four attacks during the year 1884 having been very severe—with visible injury. She was put under Dr. Stevens' care on April 22, 1887, and has been most faithful in her attendance until the present time. Insufficiency of the ocular muscles was found, and has been treated by the use of numerous kinds of prisms and by eight operations upon the muscles. During this period her average has been fifteen attacks every month, the extremes being five as the least number and twenty-two as the greatest number of attacks in a single month. There were ten very bad attacks during the year 1888 accompanied by visible injuries. The ocular treatment has, therefore, after a continuous trial of two and one half years, utterly failed to ameliorate her epileptic condition, and has been of positive harm, since the slight attacks are three times as frequent, and severe attacks are more frequent than under other methods of treatment.

24 mo. under bromides,	-	-	123 attacks.
24 " " ocular treatment,			362 attacks.

OPHTHALMOLOGICAL REPORT.

On Oct. 31, 1889—"She had abduc. 7° , adduc. 15° , exoph. 3° at 20 feet, 6° at 20 inches. She has crossed diplopia with red glass in the middle and upper field when looking to the right $V = \frac{20}{30}$."

W. O. MOORE.

CASE 3.—John McG., aet. 14; epilepsy; Dr. Stevens. This boy has had epileptic attacks, consisting of dizziness followed by unconsciousness and twitching of one extremity, never in the past eight years having had a general convulsion. He can sometimes control these attacks by running about or by putting cold water on his face. The attack occurs once or twice a day, but every month he has a remission of a week, and he occasionally has an interval of two or three days without an attack. The patient was selected by Dr. Stevens and accepted by the Commission, having been found to have muscular insufficiency. He was

operated upon four times between May 3d and Oct. 27th, 1877, during which time he had 22 attacks.

This result indicates a certain amount of improvement, the frequency of the attacks having diminished. The attacks, however, had not ceased when Dr. Stevens, on Oct. 27, 1887, reported the improvement, and no subsequent report has been made. The case, therefore, was in no sense cured.

In Dr. Stevens' report he states that during 167 days, 22 attacks have occurred, while under circumstances similar to those preceding his ocular treatment there would have been expected 167 attacks, or, on an average, one per day. To this statement an exception must be taken, since the history stated that he had intervals of two or three days without attacks and a remission of a week in every month. This is the only case in which any notes have been furnished the Secretary by Dr. Stevens.

CASE 4.—George K., æt. 13, chronic chorea, Dr. Seguin. This boy had been subject to chronic chorea for five years prior to his being sent to Dr. Stevens on May 20th, 1887. For six months prior to May he had suffered from coprolalia as well as from electric chorea. Under Dr. Seguin's care from February 25th to May 20th his condition had varied, (*and the coprolalia ceased*) there being at times a marked improvement followed by relapses. He was found to have ocular insufficiency, and during the past two years he has had twelve operations and has worn twelve different glasses. The case has not been cured or much relieved by the two years of ocular treatment. The coprolalia (*grunting*) disappeared for a time, but reappeared, and has varied in severity. Twenty-eight weeks, and again fifty-two weeks after treatment was begun, it was noted that the chorea was as bad as at first. It still persists, but at present he may be said to be improved. He now has double vision on looking to the right.

OPHTHALMOLOGICAL REPORT.

"V. = $\frac{20}{15}$; L. hyperphoria $\frac{1}{4}$ °; esophor $\frac{1}{2}$ ° in accom. 2°; abduc. 7°; adduc. 30°. Opthal. normal; fields normal; sursumduction R. $\frac{3}{4}$ °, L. 1°."—D. WEBSTER. Oct. 31. '89.

CASE 5.—Aggie H., æt. 13, epilepsy, Dr. Dana, Dr. Fisher. This girl suffered from attacks of petit mal, only having from two to four every day. She has had such attacks for two years. She was sent to Dr. Stevens on January 3d, 1888, and though orthophoria was present was thought to have "latent" hyperphoria. Operations were performed upon this patient in January and February and November, 1888, and in January, March and September, 1889; but no records have been furnished to the Secretary of the exact number of attacks. In November, 1888, she was having from three to four attacks daily, and in March, 1889, she is still having seizures every day. The mother thinks she is better.

The result of the ocular treatment pursued with regularity for fifteen months seems to be entirely negative.

OPHTHALMOLOGICAL REPORT.

May 13, 1889—"Esophor 5°, abduction 5°. She is wearing prism 1°, base out over each eye. Tenotomy of right intermus done to-day leaving orthophoria and abduction 8°."

D. WEBSTER.

CASE 6.—C. D., female, æt. 9, epilepsy, Dr. Stevens. This girl has had epilepsy, both grand and petit mal, for six years, and is feeble minded. "In the grand mal attack there are convulsive movements at first. Often the attack is ushered in by a scream. Attacks vary from two to ten minutes, and after them she is sometimes drowsy." These occurred for the past two years about once every other day, and for the past three months every day. Petit mal attacks occur also from once in a few days to many times in the same day. Dr. S. referred her to the Commission March 30th, 1888. The only note regarding this case received is a description of one operation on April 20th for tenotomy of the right superior rectus. On November 7th Dr. Stevens wrote Dr. Dana that the girl was taken in to the country in July, but that her aunt called upon him in September and said that the child was improved.

The last of definite information regarding the number

and character of the attacks prevents any conclusion as to the result of treatment.

CASE 7.—E. F., male, æt. 14, chorea, Dr. Stevens. This boy, the subject of chronic chorea for several years, was referred to the Commission on March 30th, 1888, by Dr. Stevens. The chronic movements were of the electric type, general, and accompanied by a barking sound.

No notes of the ocular conditions have been received.

On April 20th a tenotomy of the right superior rectus was made, as stated by Dr. Moore.

On November 26th Dr. Webster reports a condition of insufficiency of the muscles.

No statements regarding this case have been obtained, hence no conclusions regarding the result can be given. In a letter of Dr. Stevens, dated November 7th, 1888, he is alluded to as "the chronic boy."

CASE 8.—G. H., male, æt. 10, epilepsy, Dr. Stevens. This boy had epilepsy since the age of three, having a grand mal attack very rarely, never oftener than three in a year, last attack nine months before treatment was begun.

He also had attacks of epileptic vertigo followed by mental confusion, without any convulsive movements. These occur once in three or four weeks and are controlled by chloroform. He also has attacks of petit mal several—three or four—times daily. He was submitted to the Commission by Dr. Stevens, March 30th, 1888. No notes regarding his ocular condition were furnished. On the 12th of April and 3d of December, 1888, operations were performed upon the left and right interni respectively. A report made May 25th states that he has had no grand mal attacks, but had feared an attack of vertigo on April 27th, but had fought it off. No statement regarding the petit mal and no record of attacks is at hand. He was seen by Dr. Birdsall March 31st, 1889. He then stated from memory that he thought he had no attacks of vertigo, but that he had one grand mal attack in February, 1889. Through lack of definite data no conclusion can be drawn from this case.

CASE 9.—David S., æt. 12, epilepsy, Dr. Starr. This boy had epilepsy since the age of fifteen months, having from one to five severe and from two to five slight attacks every month under bromide treatment. He had been treated by Dr. Starr for six months. During four months (December, February, March, April) he had nine severe and fifteen slight attacks. On April 23d he was referred to Dr. Stevens, who found insufficiency of the ocular muscles. He was under treatment from April 23d to Sept. 25th, attending at least three times a week. He was operated upon several times, and wore glasses constantly. During these four months he had thirty severe and nineteen slight attacks, or an average of seven severe and nineteen slight attacks every month. At the end of this time, as he seemed to be worse rather than better, his mother refused to continue the treatment, although urged to do so. The result of stopping the bromide in April was the occurrence of nine severe fits between May 6th and May 9th. Severe fits occurred in September at the time of his father's death. But aside from these attacks the record shows that the number of fits under ocular treatment was greater than under bromide.

Oct. 31, '89.—“ *He is now under bromide treatment by Dr. Jacoby. He had three severe and two slight attacks in September; two severe and three slight attacks in October; about the same number as before ocular treatment. V. = $\frac{2}{15}^{\circ}$, abduc. 7° , adduc. 19° , esophor 4° , L. hyperphor. $\frac{1}{2}^{\circ}$.* ” STARR.

CASE 10.—Eva S., æt. 1, chronic chorea; Dr. Starr, Dr. Fisher. This girl has had chronic chorea of a mild type for three years preceding May 4, 1888, when she was sent to Dr. Stevens.

She has been in constant attendance up to the present time. Has been operated upon four times during the year and has worn glasses constantly.

The chorea has varied from time to time; has been somewhat better at times, and then again as bad as at the outset. In November, 1888, it had extended from the body and neck to the face and larynx, expiratory sounds being

quite frequent. This had not developed before and passed off after a few months.

As the result of the year's treatment there is no cure, and no marked change in the choreic condition. Her movements now, October, 1889, are about the same as at the outset.

OPHTHALMOLOGICAL REPORT.

"Eva S. has had six operations upon her eyes. She is wearing R + 2.75 D.C. axis 105°; L. + 2.75 D. C. axis 75°; and with these glasses her vision is $\frac{20}{30}$ +. R.V. = $\frac{20}{70}$ - $\frac{20}{20}$ - with + 2.25 D. C. axis 105°; L. V. = $\frac{20}{70}$ - $\frac{20}{20}$ - with + 2.50 D. C. axis 75°. Orthophoria with or without her glasses; abduction 6°; adduction 36°. Opthal. no lesion observed. No diplopia."

WEBSTER.

CASE II.—Miss L. P., æt. 15; chronic chorea; Dr. Stevens. This girl was both choreic and hysterical, the irregular movements being very marked and her mental irritability and instability evident. She is subject to hysterical fancies. Her motions are partly choreic, partly wilful.

She was submitted to the Commission on July 5, 1888. No records of the eye conditions of treatment, or of present condition have been received. A letter from Dr. Stevens, dated November 7th, 1888, indicated that she was still under treatment at that time.

Dr. Webster saw the patient at Dr. Stevens' office, May 31st, 1889. She was operated upon several times for hyperphoria and the condition of exophoria is now present; she still had slight choreic movements, but said that she was much better. It seems that she had been subject to incontinence of urine—a fact not mentioned in her history—which had made her life miserable. This condition has ceased during the past year so that she now enjoys life. Present condition of eyes unknown.

CASE 12.—E. W., æt. 24; epilepsy; April 25, 1889; Dr. Dana, Dr. Starr. She began to suffer from epilepsy at the age of 19. Slight seizures increased in frequency; until October, 1888, she was having 2 or 3 daily. During the

summer of 1888 she had 4 general convulsions. Under bromide treatment she was having petit mal about 2 or 3 times every two weeks, usually at time of menses.

She was found to have very marked insufficiency of the ocular muscles and was accepted by Dr. Stevens as a good case in April, 1889. During the summer four operations have been recorded.

At present (October 25) she says that she has had one severe fit during the summer (viz., May 23d) and is now having and has had two or three attacks of petit mal monthly. She is therefore improved.

OPHTHALMOLOGICAL REPORT.

Nov. 4.—“She has been operated upon nine times. R. V. $\frac{3}{30}$ with — 4 D.; L. V. $\frac{2}{30}$ with — 9 D. Hyperphor. 0° , exophor. 2° , with her myopia corrected. Esophoria 1° . Ophthal. exam. large staphyloma posticum in both eyes.”

D. WEBSTER.

CASE 13.—Mary McK., æt. 31; epilepsy; Dr. Dana. Patient had migraine from age of 12 and epileptic seizures of petit mal type since age of 22. She had been having two to three such attacks monthly when first seen in March, '89, by Dr. Dana. She was reported to the Commission April 25th, having had 8 attacks in April. She was found to have insufficiency of the ocular muscles. Bromides were stopped May 1st and during the summer she has been operated upon several times. She said (September 10th) that she was better and says her attacks are lighter. The record, however, shows that she had on May 9th, on June 12th, on July 22d, attacks of which 3, 4 and 2 respectively were severe. On August 1st, severe attack, on the 19th, the last recorded. During September she had four, during October she had 5 attacks of petit mal.

It is evident that she is now having fewer attacks than she had under bromide treatment in April. The number of attacks, however, is about the same as when she first applied to Dr. L. Dana, viz.: three to four monthly.

CASE 14.—Agnes H., æt. 31; epilepsy; Dr. Birdsall. This patient has had severe epileptic attacks for ten years

at long intervals, and slight attacks frequently. Thus, on Jan. 9, Feb. 6, March 11 and April 15, slight attacks occurred, and a series of seven severe attacks within four days in April, during a cessation of bromide treatment. She was referred to Dr. Stevens on April 25th. She was found to have an insufficiency of the ocular muscles, and has been under treatment up to the present time (Oct. 22nd). She had 29 attacks in May, 20 in June, 30 in July, 24 in August, and says that she has had one every day for the past month. During these five months she has also had four severe fits.

Her general condition is about the same. Her attacks are more than twice as frequent as under bromide treatment, and no improvement has been noticed during the past month.

OPHTHALMOLOGICAL REPORT.

"R. V. $\frac{3}{20}$ + in each eye but sees $\frac{1}{10}$ with both eyes at once with + 1 D. accepts no glass with either eye singly. Hyperphor. 0° , esophor. 1° to 2° in accom. exophor. 9° , abduc. 5° , adduc. 27° . Sursumduction, R. 1° , L. 1° . Ophthal. exam. shows pale temporal halves of discs. Visual field normal. No diplopia."

D. WEBSTER.

The following are summaries of the histories of the cases submitted to Dr. Stevens and subsequently withdrawn, with the reason for withdrawal.

These are the cases not counted according to the plan.

CASE 15.—Elizabeth K., aet. 25; epilepsy; Dr. Dana. This patient had suffered from attacks of petit mal since the age of eleven, and from grand mal since the age of thirteen. She has had several fits every week for two years, and occasionally suffered from a series of fits followed by a period of mental aberration. Under Bromide and pepsin she had been having from one to three attacks of grand mal every week.

She was referred to the Commission, on April 6th, and found to have insufficiency of the ocular muscles. She was operated upon by Dr. Stevens, on April 18th. She was

very irregular in her attendance, not being able or willing to follow up the treatment with regularity, and therefore, after one month, was withdrawn by mutual consent.

No data are in possession of the secretary regarding her further history.

CASE 16.—Stephen W., æt. 17, Epilepsy, Dr. Dana.—This boy had right hemiplegia with aphasia at the age of four, slight traces of the former remaining. He developed epilepsy at the age of thirteen, the spasms being at first mulateral, but after a year becoming general. Without bromide he rarely goes more than two or three days without an attack.

The epilepsy was thought to have an organic basis in a cicatrix in the left cerebral hemisphere, but in spite of this statement Dr. Stevens accepted the case and it was put under his care on May 19th, 1887.

Occular insufficiency was found, and he was operated upon twice during June by Dr. Stevens. In October, however, having had eleven attacks every month during the summer, and not being improved, Dr. Stevens requested that he be withdrawn and not counted in the cases on the list. This was therefore done.

CASE 17.—Mary L., æt. 30, Epilepsy, Dr. Stevens.—This girl, a servant suffering from nocturnal epilepsy, was submitted to the Commission by Dr. Stevens and accepted in May, 1887. It was found, however, that she could not attend with sufficient regularity and her case was therefore dropped from the list.

No notes have been furnished to the Secretary regarding her original condition, as she was seen but once by Dr. Stevens.

CASE 18.—John C., æt. 21, Dr. Seguin.—This patient who had suffered from epilepsy since a sunstroke at the age of nine, had attacks of grand mal about once in two weeks. He was sent to Dr. Stevens on May 20, 1887, and all medicine was stopped. He was found to have insufficiency of

the ocular muscles, and an operation was performed on June 6th. This operation was followed by a series of severe convulsions, thirty-one occurring within a month. In view of these attacks the patient and his friends expressed unwillingness to go on with the treatment, and he was withdrawn from Dr. Stevens care on July 25th.

After resuming bromide treatment he had but one attack in August, and but one in September. In October he had access of attacks, twenty occurring in two days while on a visit to Boston, but he had none between October 3d and January 1st, 1888.

Dr. Seguin believes that the status epileptus produced by the cessation of medical treatment would probably have proved fatal had bromides not been resumed in July.

CASE 19.—Patrick H., æt. 22, epilepsy, Dr. Dana.—This patient had epileptic seizures, both grand and petit mal, daily, though at times he would go a week without an attack. He was sent to Dr. Stevens September 20th, 1887, and found to have insufficiency of the muscle of the eye.

He was operated upon twice during October, 1887, and on November 4, 1887, was found by Dr. Moore to have no insufficiency. He discontinued going to Dr. Stevens during December and January, but on February 22d a third operation was performed. The record of attacks during October, November and December is imperfect or lost. During January, 1888, he had twelve attacks of petit mal, four attacks grand mal. During February, 1889, six of petit, five of grand. During March, fourteen petit, three grand. On April 2d patient said he had fewer attacks than before the treatment was begun, but circumstances prevented his continuing attendance. From April to December, 1888, his fits were somewhat less numerous than before treatment, but the change is slight. He may be said to be improved.

CASE 20.—John S., aet. 38; epilepsy, Dr. Dana, Dr. Nilsen. This patient had nocturnal epilepsy, one attack every month of grand mal type, from December, 1855, to May, 1887, and two attacks per month in August, September and October, 1887.

He was sent to Dr. Stevens on October 30, 1887, and muscular insufficiency was discovered.

No records of treatment have been furnished, and probably none was pursued, for the patient could not attend regularly, and his name was therefore withdrawn. It is interesting to learn that under bromides and oxide of tin he has now March, 1889, gone a year without any attacks.

CASE 21.—Jennie P., aet. 25 ; epilepsy, Dr. Seguin. The patient had suffered from epilepsy for several years, having both grand and petit mal attacks.

She was sent to Dr. Stevens December 20th, 1887, and was found to have ocular insufficiency. She was operated upon three times. Her attendance having been irregular, she was withdrawn within a month by mutual consent.

The history of this case is imperfect. It is in the possession of Dr. Stevens, and no copy having been taken and access to the history having been refused by Dr. Stevens, all data are wanting.

CASE 22.—John D., aet. 29 ; epilepsy, Dr. Dana, Dr. Leszynsky. This patient had grand mal and petit mal attacks. He was referred to the Commission on March 1, 1888, and was found to have an insufficiency of the muscles, for which prisms were given. An operation was appointed for April 6th. No notes of this are at hand. But on April 30th Dr. Stevens requested that he be withdrawn from the list, his social relations being not such as to make him a good subject for a test case.

CASE 23.—A. B., male, aet. 16 ; epilepsy, Dr. Stevens. This patient who had had epilepsy since the age of one year, was referred to the Commission by Dr. Stevens on February 28, 1888. The history submitted gives no account of the number or character of the attacks, of the conditions found in the eyes or of any operations performed. A note of Dr. Birdsall states that on May 28th the notice says that the attacks have been less frequent. He was taken abroad in July, and his failure to return led Dr. Stevens to withdraw the case from the Commission on February 27th, 1889.

CASE 24.—Alfred B., aet. 26; epilepsy; Dr. Dana, Dr. Jacoby. The patient had well marked epilepsy, with attacks of grand mal occurring from three to four times every month, and attacks of petit mal, both day and night, from five to seven daily.

Under bromide the severe attacks had been reduced in number to one or two a month, and the petit mal had been controlled at one time by nitro-glycerine for fifteen months, in 1884-85. He was referred to the Commission on April 10, 1888, and examined by Dr. Stevens, who found an insufficiency of the ocular muscles. Two operations were performed April 13th and May 4th, and glasses worn constantly until the latter part of June, 1888. He had one grand mal attack in April; four in May; three in June, and his attacks of petit mal did not diminish. In June he discontinued attendance voluntarily and refused to return. Between July 1st and December 1st he averaged three attacks of grand mal monthly. No improvement was produced by the treatment, and he was not counted, as he withdrew within three months.

CASE 25.—Adolph S., aged 23; epilepsy; Dr. Dana, from Dr. Leszynsky. This patient had petit mal attacks at the age of eleven, and grand mal attacks at the age of twelve. At the present time he has a general convulsion about once a month and attacks of petit mal every week. During February, 1888, under bromide treatment he had one grand mal and six petit mal attacks.

He was referred to Dr. Stevens on February 29, 1888.

No records of the ocular conditions have been furnished. The patient was operated upon once, but was so irregular in his attendance that he was withdrawn soon after by mutual consent. A letter from Dr. Leszynsky, dated April, 1889, contains the statement that the patient is still visiting Dr. Stevens, but is not in any way improved; his mother reporting that his attacks are becoming worse.

CASE 26.—Mrs. Q., aged 26; epilepsy; Dr. Dana. This patient, suffering from a very severe type of grand mal, with

several daily attacks, was sent to Dr. Stevens, November 24, 1888, but was at once withdrawn because of his objection to the patient, who was unpresentable and had several attacks in his office.

CASE 27.—Tillie L., aged 18; epilepsy; Drs. Jacoby, Starr, Dana. The patient had suffered from epilepsy from the age of twelve, and for the past two years had been having about two attacks every month, all recent attacks having been nocturnal. They are of the grand mal type.

The patient was at work, and could not afford to attend with sufficient regularity, and therefore, by mutual consent, was withdrawn.

CASE 28.—Anna B., aged 29; epilepsy; Dr. Seguin. This patient developed epileptic attacks at time of first menses, and these have continued up to date. She has been under treatment since 1880 at intervals, and presents no other symptoms than those of epilepsy; no evidence of cerebral or spinal disease.

She has from two to five attacks monthly.

She was sent to Dr. Stevens on April 25th, but rejected by him on the ground of the existence of optic-nerve atrophy, visual field in one eye contracted one-half. Hence withdrawn.

SUMMARY OF THE WORK OF THE COMMISSION.

Total number of patients examined by the Commission, and sent to Dr. Stevens for examination.	-	28
Suffering from epilepsy,	- - - - -	23
“ “ chronic chorea,	- - - - -	5
Number of cases found to have ocular insufficiencies,	-	28
Number of cases withdrawn without any treatment,	-	5
“ “ “ within 1 month after exam.,	3	
“ “ “ “ 2 months “ “	-	1
“ “ “ “ 3 “ “ “	-	1
“ “ “ “ 4 “ “ “	-	2
“ “ “ “ 5 “ “ “	-	1

REASONS FOR WITHDRAWAL OF PATIENTS.

Inability of patient to attend regularly,	-	-	-	5
Declined by Dr. Stevens on ground of organic disease,				2
Mutual consent of Dr. Stevens and member sending case, for satisfactory reasons, such as non-attendance,	-	-	-	5
Discontent of patient because of increase of symptoms,				3
Number of cases continuing treatment for four months or over,	-	-	-	15

DURATION OF TREATMENT IN THESE CASES.

4 months,	-	-	1	12 months,	-	-	1
5 "	-	-	5	18 "	-	-	1
6 "	-	-	1	21 "	-	-	1
8 "	-	-	1	29 "	-	-	1
10 "	-	-	1	30 "	-	-	2

It will be seen from the summary that of the twenty-eight cases sent to Dr. Stevens but fourteen are available for any conclusions, the others having been withdrawn for some reason, and therefore not being counted according to the Plan of Proceedings.

The following table contains numbers of the cases under treatment continuously for a period exceeding four months; the duration of treatment in each case; the date at which the patient was sent to Dr. Stevens; the date of the last report of the condition of the patient, and a statement of the apparent result of treatment.

For details regarding each of these fourteen cases the Commission has appended the full history of each case. (*Vide Exhibit F.*)

Case.	Duration of Treatment.	Date of First Visit.	Date of Last Report.	Result.	Result.*
1. Chorea.	30 months.	April, '87.	Oct. '89	Improved.....	
2. E	30	April, '87.	Oct. '89.	Not Improved..	
3. E	5	May, '87.	Oct. '87.	Improved.....	
4. Chorea.	29	May, '87.	Oct. '89.	Slightly Imp'd.	Improved.
5. E	21	Jan. '89.	Oct. '89.	Not Improved..	
6. E	4	Mar. '88.	July, '88.	Unknown.....	
7. Chorea.	8	Mar. '88.	Nov. '88.	Not Improved..	
8. E	12	Mar. '88.	Mar. '89.	Unknown.....	Improved.
9. E	5	Apr. '88.	Sept. '88.	Not Improved..	
10. Chorea.	18	May, '88.	Oct. '89.	Not Improved..	
11. Chorea.	10	July, '88.	May, '89.	Not Improved..	Improved.
12. E	6	Apr. '89.	Oct. '89.	Improved.....	Slightly Imp'd.
13. E	5	May, '89.	Oct. '89.	Not Improved..	
14. E	5	May, '89.	Oct. '89.	Not Improved..	

* Corrections only noted.—Ed.

A summary of these 14 cases shows :

Total cured,	-	0	
Total improved,	5		One very much improved.
Total unimproved,	8		
Total unknown,	1		

A summary of these 14 cases shows :

<i>Total cured,</i>	<i>-</i>	<i>0</i>	
<i>Total improved,</i>	<i>6</i>		<i>One very much improved.</i>
<i>Total unimproved,</i>	<i>7</i>		<i>Two slightly improved.</i>
<i>Total unknown,</i>	<i>1</i>		

	Total cured	0	
C. Chorea, 5.	Total improved	5	3 Chorea, 2 Epilepsy.
	Total unimproved	8	2 Chorea, 6 Epilepsy.
E. Epilepsy, 9	Total unknown	1	Epilepsy.
	<i>Total cured</i>	<i>.....</i>	<i>0</i>	
C. Chorea, 5	<i>Total improved</i>	<i>.....</i>	<i>6</i>	3 Chorea, 3 Epilepsy
	<i>Total unimproved</i>	<i>.....</i>	<i>7</i>	2 Chorea, 5 Epilepsy
E. Epilepsy, 9	<i>Total unknown</i>	<i>.....</i>	<i>1</i>	Epilepsy.

It will be seen that five (*six*) of the patients are at present improved. These are :

Case 1.—Eliz. C.,	-	Chorea,	-	Dr. Seguin.
" 3.—John McG.,		Epilepsy,	-	Dr. Stevens.
" 8.—G. H.,	-	Epilepsy,	-	Dr. Stevens.
" 11.—Miss L. P.,		Chorea,	-	Dr. Stevens.
" 12.—Gertrude W.,		Epilepsy,	-	Dr. Dana.
" 4.—George K.	-	Chorea,	-	Dr. Seguin.

In regard to (1) the first improvement took place thirty weeks after treatment was begun, and the condition has not been one of constant improvement, but of considerable variation in the choreic state. At times the girl has been better, at times worse. On October 23, 1889, she was free from chorea. *At no time has she been free from chorea for one month.*

In regard to (3) the epileptic attacks were of the nature of vertigo, the patient never having had a convulsion. He was able to control these attacks at times by running about,

or by applying water to the face. The history states that the attacks occurred once or twice daily, but that every month there was an intermission of a week, and occasionally he had two or three days without an attack. He had no attacks for a week before treatment was begun. No exact record of attacks before treatment, is given. During five months of treatment he had twenty-two attacks, and the attacks had not ceased when, in October, 1887, Dr. Stevens reported improvement. No further report has been obtainable.

In regard to (8) the patient had epileptic attacks of several kinds—a general convulsion once or three times yearly, epileptic vertigo once in three or four weeks, and petit mal several times daily. When last seen he had had one convulsion after a year of treatment, and he stated from memory that he had had no attacks of vertigo for some time. He made no statements regarding petit mal attacks, of whose occurrence he was ignorant. No records have been obtainable and hence no exact conclusion can be drawn, excepting one of general improvement (*in respect to attacks of vertigo*).

In regard to (11) the girl had chronic chorea, was very hysterical, and had incontinence of urine which made her life miserable. During the year of treatment her hysterical condition improved; the incontinence ceased, and with its cessation her life became more enjoyable, and her general condition improved. The choreic motions continued at the time of the last report, but were less than at the beginning of treatment.

In regard to (12) she is having about as many attacks as she had under bromides, but far less than when she had no treatment whatever.

In regard to (4) the chorea is only somewhat less than at the outset.

It will be noticed that three of the five cases reported as improved were presented to the Commission by Dr. Stevens, being his private patients, and that no exact data have been obtained regarding them. This is especially to

be regretted as the exact degree of improvement cannot be ascertained.

In regard to (6) also a patient of Dr. Stevens, no data have been furnished as to the result.

It will be seen that eight of these patients are unimproved. These are :

CASE 2,	-	Flora K.,	-	Epilepsy,	-	Dr. Starr.
" 4,	-	George K.,	-	Chorea,	-	Dr. Seguin. ⁹
" 5,	-	Aggie H.,	-	Epilepsy,	-	Dr. Dana.
" 7,	-	E. F.,	-	Chorea,	-	Dr. Stevens.
" 9,	-	David S.,	-	Epilepsy,	-	Dr. Starr.
" 10,	-	Eva. S.,	-	Chorea,	-	Dr. Starr.
" 13,	-	Mary McK.,	-	Epilepsy,	-	Dr. Dana.
" 14,	-	Agnes H.,	-	Epilepsy,	-	Dr. Birdsall.

The patients with chorea (4,¹³ 7, 10) remain about in the condition in which they were when sent to Dr. Stevens. They have been at times slightly better and at times much worse than they are at present. The treatment has had apparently no influence on the course of the disease.

The patients with epilepsy have continued to have attacks. In all of these cases the immediate effect of withdrawing bromides was to cause an increase in the number and in some cases in the severity of the attacks. This rarely continued longer than three months. Then a fairly regular recurrence of attacks is noted. In some cases twice or three times as frequent as under bromide treatment.

The investigation therefore demonstrates incidentally the value of bromide as contrasted with non-medicinal treatment.

Nor can any special connection be traced in these cases between the occurrence of attacks, and changes in the ocular conditions produced by operation or by change of glasses. There has been no apparent result in these cases except that the cessation of bromide treatment has allowed the disease to take its course.

¹³ Added to corrected list of improved.—ED.

It may be mentioned, however, that many of these patients say that they are better in spite of written records proving the increased frequency of attacks. This is not wholly due to an increase of hope and confidence in the success of the treatment, but is to be ascribed in part to the freedom from the depression of mind and of bodily functions produced formerly by the use of bromides.

Other of these patients have complained much of headache and vertigo, which have appeared during the treatment (*and in several a condition of diplopia has been produced and now remains*).

And a few of these patients have been made exceedingly miserable by the great increase in frequency and severity in the attacks, which has finally necessitated an abandonment of the ocular treatment and a return to drugs. This statement applies to a number of the cases, withdrawn and not counted in the list.

In view of these facts, your Committee cannot but express the opinion that, so far as this investigation has warranted a conclusion, the method of Dr. Stevens does not afford a sufficient degree of relief to patients suffering from chorea and epilepsy to warrant its adoption or recommendation to the members of the Neurological Society, as a means of cure, or as the sole therapeutic measure. It will be noticed that none of the cases have been cured—all remain indefinitely under treatment; a perfect permanent ocular equilibrium not having been secured in any case. If it is impossible to secure such equilibrium, a cure is not to be expected, granting the theory of Dr. Stevens. If it is possible to secure such equilibrium, it is to be supposed that it can be secured within at least a year (*two years*). Yet four of these cases have been under constant treatment for two and one-half years without this result.

There is, however, a third possibility which the Commission desires to present to the Society. And this is, that the insufficiency of the muscles may be merely one of the conditions in a class of diseases, in which the muscles are deeply involved, and one of which class epilepsy and chorea are examples, and that the tone of the ocular muscles may

vary with that of the other muscles of the body in accordance with the general condition of the patient or of the special strain, such as may occur in a spasm. It is also possible that the tonicity of the ocular muscles is impaired by the long-continued use of the bromides.

It may be admitted, that great insufficiency of ocular muscles, like myopia or hypermetropia, should be corrected.

If this possibility be approved, it becomes evident that treatment directed to an effect of the malady and wholly without relation to its cause, will necessarily be unsatisfac-

ERRATA.

Page 677, line 11 from bottom, for "on May 9th, on June 12th, on July 22d attacks" read *in May, 9; in June, 12; in July, 22 attacks.*

Page 678, line 1, for "on Jan. 9; Feb. 6," &c., attacks, read *in Jan., 9; Feb., 6, &c., attacks.*

Page 685, line 9, *Two slightly improved* refers to the improved, and not to the unimproved cases.

Page 667, foot note 11, insert the word *all* before the words "*drop out.*"

M. ALLEN STARR,

Secretary of the Commission.

REPLY OF DR. STEVENS

TO THE

REPORT OF THE COMMISSION TO EXAMINE
THE PROPOSITION THAT FUNCTIONAL NER-
VOUS DISEASES ARE LARGELY DEPEND-
ENT ON OCULAR DEFECTS.

To the Neurological Society:

Contrary to the agreement and stipulations between the Commission appointed by this Society and myself, the Commission has resolved to submit its report at this time. The importance of the subject of the inquiry which it undertook is sufficiently great to have warranted not only a fair, but even a generous interpretation of the conditions agreed upon for making this report.

The Commission, in making its report at present, not only violates the letter of our engagements, but apparently disregards what should have been the aim of the inquiry—a united effort to discover the truth. It violates also both letter and spirit of the engagements in that, whereas this investigation was instituted and carried on not to establish a part, but the whole of a doctrine, the Commission, in disregard of its pledges and of the plain meaning of the agreement, advertises and presents a report upon two forms of disease; thus leaving the subject of investigation as unsettled as before—a contingency against which I entered my earnest protest before entering upon the work of the investigation, and against which I accepted the formal pledge of the Commission. The challenge sent to me in the name of this Society was a challenge to investigate a proposition, and these two forms of disease were accepted on the pledge that the result should represent that doctrine.

Protesting against this whole action, which is contrary to the interest of medical science, as well as a personal in-

justice, it is my purpose only at this time to call the attention of those interested in the inquiry to the spirit in which it has been prosecuted by the leaders of the Commission and the errors with which the report abounds.

In order to obtain a just conception of all the conditions which have surrounded this inquiry, we must go back to the period at which it originated. It does not reflect unfavorably upon the sincerity or candor of any member of the Commission when the fact is recalled, which was at that time notorious, that a number of the members of this Commission had committed themselves against the views included in the proposition presented to this Society in my paper of March 1st, 1887, long before the paper was read, and in several instances by public denunciations. It is also a notorious fact that the opposition to these views had, to a considerable extent, among the members of the profession, taken the form of personal antagonism to the advocate of that proposition.

When, therefore, this Society, through its representative, requested me to enter upon this investigation, it virtually invited me to enter a hostile camp and to trust to the fairness of those who were known to be opponents.

Whether a consent to enter upon a trial of great importance to the truth which I advocated, as well as my own interests, was, under such forbidding circumstances, wise or prudent, is a question upon which it is not now necessary to enter. The facts, however, may throw some light upon the subsequent position of the two parties engaged in the investigation.

It is proper in this connection to express my sincere appreciation of the uniform courtesy and kindness of the two oculists associated with the Commission during the whole period through which the investigation has continued, and to say that with some other members of the Commission my relations have been only of a pleasant character. Whatever their position may be in respect to the endorsement of the report presented, I entertain for them only sentiments of friendly respect.

Responding to the formal challenge of the President of

the Society, I offered a plan upon which I believed that an inquiry could be made of interest and value to the medical profession and which appeared entirely just to all engaged. This plan the President in a letter declared to be "perfectly just and fair", and added his assurance that all were anxious to be entirely just to me. No sooner, however, had my acceptance to the proposition been received than the Commission, which had already been appointed, demanded radical changes, and several weeks were spent in urging and making alterations of this plan which should give the greatest possible latitude to the Commission with the severest restrictions upon the one who was to perform the labor and accept the responsibility. The vexations and annoyances of those weeks of preliminary work would have deterred anyone not possessed of the highest conviction of the truth and dignity of his cause from further relations with the Commission.

The work of the investigation, however, at length commenced, and it was seen that the Commission which had taken so many weeks to reconstruct the plan in such a manner as to be more binding upon me, was paying slight heed to the obligations of that plan themselves. It became further evident that it was the purpose of the Commission to send for treatment not only the most incorrigible cases, but apparently to keep the cases down to the lowest possible number. It is this last seeming purpose which has, through a long period, proved a source of vexatious controversies. For notwithstanding the fact that after a year had elapsed and only nine cases had been sent by the Commission, including two that were wholly unfit, and one that had never made but a single visit to my office, the Commission had already made repeated demands that a report be rendered.

Thus, while the provisions of the agreement required that the report should be based upon a number of suitable cases, not less than twelve nor more than twenty, the Commission was demanding that the report should be made upon five cases sent by them and one furnished by myself. But even of these six not all had continued under treatment ; in

fact, only four cases sent by the Commission were under treatment when one of these demands was made.

Even as early as April, 1888, the Chairman of the Commission in again urging a report, declared that it was impossible for the Commission to obtain cases and that therefore as the inquiry had continued for a considerable time, a report was due to the Society. To this my reply was that the Commission had issued the challenge for this inquiry, and that it should be easier for a large Commission to furnish twelve suitable cases than for one man to cure them.

Again, in this report the Commission returns to the familiar and disheartening complaint that an appeal made by it in April, 1888, by postal cards sent to all the members of the Society to aid them in the work of obtaining the necessary cases, "proved almost useless."

Again and again has this question arisen, in every instance in violation of the letter and spirit of the agreement.

When, in April last this ever recurring subject presented itself, upon my instance that the Commission must first comply with the rules of the agreement, one of the members of the Commission visited me and inquired what would be satisfactory to me, to, to which I replied that a literal compliance with the engagements would satisfy me. He then inquired if I would consent to a report in November in case the Commission would, within a month, send four new and suitable cases for the inquiry. To this I replied that if these patients continued under treatment, I would do so. He objected that this might cause indefinite delay. I at length agreed that should four suitable cases be sent, and should these continue until they could be considered fairly under my treatment, I would be responsible for retaining them and would not interpose objections if some of them should discontinue. I did not, as the Commission reports, "request" four patients. I accepted the proposition offered, on certain conditions. Nor have I ever directly or indirectly, as represented by the Commission, "requested" four or any number of cases. Nor have I, as stated by it, "com-

plained" because many cases were not sent to me. I have declined to comply with its demand to surrender the terms of the agreement, and I submit it to the judgment of this Society and the public whether it is either dignified or just for this Commission to represent that I have been a seeker for favors at its hands. The member of the Commission already mentioned a few days after this interview reported, as the Commission's representative, that there would be sent four suitable cases within the next four weeks, and that on the basis of this agreement a report was to be made in November. Written statements of officers of the Commission confirmed this oral agreement. Three cases came and were accepted. Another came, but as she not been informed that she was to discontinue all drugs, she declined to attend. Of this the Commission was at once notified, and at the end of the month two notices were sent, stating that only three cases had been received. To these letters there was no response. Autumn came and as usual there was a demand for a report. To my objection that the Commission had failed to do as it had agreed, it was said that inasmuch as more than one of the cases sent had remained, the agreement was virtually complied with. This was certainly not a correct claim. It was my privilege to demand a compliance with a specific stipulation which had been made to me, as I had reason to suppose, in good faith, without giving any reasons for such a demand. There were good reasons, and some of these were submitted to the Commission.

The Commission declares that a letter from me at this time contains two distinct propositions. "One states that he (Dr. Stevens) would like four more acceptable cases; the other that one or more of the four patients continue under treatment till November." Permit me to say in the most emphatic manner that no such letter has been written by me, and that the Commission has no such letter in its possession, and this is not an exceptional instance of the practice of the Commission in this report of making quotations purporting to be from letters from me, but which letters never existed.

The Commission complains at great length that reports were not sent to it. No notes, except such as were sent, were required by the terms of the agreement, and we may properly inquire why so great impatience was experienced by the Commission, because all my notes of the cases were not placed at its disposal. The Commission itself explains the reason for that anxiety when it says in the report just read: "During the Autumn of 1888, as numerous cases had been under treatment by Dr. Stevens for many months, it was discussed at several meetings when a final report should be made. Preliminary to the planning of a report it was necessary to collect all the data relative to the cases." . . . "This was made difficult by the refusal of Dr. Stevens to furnish his notes of the cases."

The reason, then, why the Commission did not render the final report a year ago, appears not to have been respect for the agreement into which it had entered, but the difficulty of obtaining the materials for making the report before it had furnished the materials for doing the work.

It will help to a clear understanding of the character of the notes required and of the impropriety of the demand, if we examine a letter of this period when the Commission had again determined to make its final report. This letter, dated October 7th, 1888, reads as follows:

"I am directed by the 'Stevens Commission' of the Neurological Society to request you to send me copies of your records of the cases submitted to you by the Commission in accordance with Section 5 of the plan of proceedings. The Commission desires to make a report in December, if possible; but it is essential that your report of the condition found, of the operations done, and the subsequent condition and of the present state of each case, should be in their hands before such report can be prepared. Will you kindly state which of the following patients have been treated and which of them are still under treatment?"

(Here follows a list of names.)

Yours very truly,

(Signed) M. ALLEN STARR, *Secretary.*"

From the above it appears:

First, that the Commission had resolved to report the results of the investigation; and second, that it demanded from me a complete report of all the original conditions, the treatment, progress and present conditions of all the patients. In other words, it demanded a complete and final report.

Section 5 of the plan, in accordance with which this report was said to be demanded, distinctly refers to the final report, and if there could be a doubt as to the meaning of this section it should be interpreted by the corresponding provision in the plan originally submitted by me, in which provisions for the final report of the Commission and of myself are contained in the same section, and the meaning is explicit. There is nowhere in the articles of agreement any provision for the kind of report called for in the above letter, except that which is to be made at the close of the investigation. This Section 5 of the revised plan reads: "A report shall be made by Dr. Stevens to the Commission of the conditions found, of the methods and details of treatment, and the progress of the case and the conditions remaining at the times of making the report."

Will any right-minded man claim that this section calls for a report such as is demanded in the above letter, to be made by me at any time at the will of the Commission? Or will any reasonable person withhold his approval of my refusal to furnish such a report when the demand for it was coupled with the announcement of the Commission that it was to use this material in a flagrant violation of its own obligations to the public and to me?

This Commission, representing a large society, had publicly challenged me to an investigation, to be based upon not less than twelve nor more than twenty cases. Now, after a notable failure on its part, shown by the fact that after the first nineteen months of its existence, this Commission, which had so boldly challenged me, had sent to my office, including those who came but once, the syphilitic and traumatic, the intoxicated, the cases of gross cerebral lesions, and others which were not in the inquiry, according to the plan—in all fifteen persons, of whom only seven were

then under my charge. The Commission was perfectly informed of this state of facts, and the repeated announcements of its purpose to report were in absolute defiance of the provisions of its own plan.

I freely confess that the annoyances of this ever-recurring conflict over a subject concerning which there could be no reasonable question of the right, have been to me a source of greater trouble than the treatment of these unfortunate cases during all this time.

In the lengthy presentation of its case in respect to the difficulties claimed to have been encountered by the Commission in obtaining records, a great deal of matter is included which does not accord with the evidence in my possession. The report asserts that "Dr. Stevens has not furnished the Commission with reports of the case, or copies of his various notes of them (or allowed of copies being made at the Commission's expense, as was proposed.)"

How this assertion harmonizes with the facts may be seen from the following statement :

May 22d, 1887, the Secretary of the Commission, wrote, asking if Dr. Stevens would "allow such a copyist as Dr. Foster may select to copy the histories already in your possession," etc. To this an affirmative reply was sent. As Dr. Foster did not send the copyists, my own clerk prepared the copies of all cases then under treatment, and sent these copies to the Secretary, without expense to the Committee. The Secretary acknowledged them in the following letter :

24 West 48th Street, Oct. 8th, 1887.

Dr. GEO. T. STEVENS.

Dear Doctor:—The report of the six cases are received. Will you kindly let me know what the expense of copying has been, and I will refund you. Will you also kindly inform me how many cases have dropped off and do not continue to come and to whom those cases belong, so that the doctors can look them up and send them back ?

Yours very truly,

M. A. STARR.

It will thus be seen that the assertion of the Commission in its report that I refused to allow copies to be made is incorrect. The statement also that Dr. Stevens has put the Commission to serious inconvenience by retaining the only existing notes of one case can be positively disproved by the written testimony of the Chairman of the Commission.

I have prolonged this portion of my statement to this very considerable extent, because the Commission has expended great pains and much space to make it appear that in refusing to comply with its demands I was violating the spirit and letter of the compact. I reply that I resisted the declared intent of the Commission to disregard the plan which it had offered, and I think there are many who will approve of that resistance.

The Commission also complains that Dr. Stevens declined to make new stipulations respecting the time during which patients should continue under treatment. To this and similar propositions I uniformly replied that it would be better to comply with the present agreement than to make new ones.

In all this matter, then, in which the Commission labors through many pages to produce the impression that Dr. Stevens was an obstacle to the work of the Commission, it appears that Dr. Stevens simply declined to consent to a violation of the original contract or to resort to a new one to suit the purposes of the Commission.

Before passing to a review of the statements of the Commission relating directly to the conditions of the patients before and after treatment, it is desirable to look further into the general spirit and motive of this report.

One of the sections of the plan submitted by me to the President of this Society in March, 1887, reads as follows :

"8th. Should any patient decline or neglect treatment without consent either of a majority of the Commission or of myself, such case should not be regarded as in the inquiry."

In the plan, as revised by the Commission, a part of section 5 reads as follows:

"If any patient declines or neglects treatment, such case shall not be regarded as included in the inquiry, or counted."

These two forms of statement are clearly intended to mean that the report of the inquiry is to be confined to those cases only which come within the proper meaning and scope of the inquiry. Why, then, does this report contain long details of thirteen cases, which, according to the stipulation, were "not to be regarded as included in the inquiry or counted"? Can it be that, by presenting this long array, representing some cases which came once only, and others which should never have been sent at all, the Commission desires to convey the impression that there has been a much larger amount of good material furnished than has been used to good purpose? Surely no scientific purpose can be served by this array of imperfectly reported cases. If the motive is not to place Dr. Stevens in a false light, the plan has been unfortunately conceived. These thirteen pages of type-written material, inserted in this report in direct defiance of the articles of agreement, must surely have a deeper significance than the mere setting forth that a Commission of this Society has been able to find a certain number of epileptics and choreics, some of which had come to me but a single time, of which most were unfit and all were out of the inquiry. Whatever the purpose of inserting these cases in the report may be, the evident effect is to cast a reflection of failure upon Dr. Stevens. It is also in disregard of a specific promise of one of the Commission that, if I would send him copies of cases not in the inquiry, they should not in any way be included in this report. The copies were sent, and the cases are in.

But there are details in these and other cases which are, if the Commission is not aiming to induce prejudice in the minds of the reader, certainly unfortunate. For instance, John D., Case 22, was a case of old syphilis with optic-nerve atrophy and with a fresh wound measuring several inches

in length, extending through the thickness of the scalp, an injury inflicted by a flat-iron in the hands of his wife. These facts were stated in a letter to the Secretary declining to accept the case. He is in this report stated to have been rejected on account of his "social relations."

Mrs. Q., Case 26, was a respectably dressed and well-behaved German woman, who was no more objectionable, from the standpoint of her appearance, than others sent by the Commission. She was a case in which optic-nerve atrophy was only one of many indications that the patient was suffering from extensive organic degenerations. For these reasons solely, Dr. Webster, and afterward Dr. Dana, fully concurred with me in the view that she was an unsuitable case. The report says: "She was withdrawn because of his objection to the patient, who was unpresentable and had several attacks in his office." The patient was neither unpresentable, nor did she have several attacks in my office.

What motive has induced the Commission to apply reasons, which were not the real ones, for the withdrawal of these patients? Was it to convey the impression that they were rejected for slight causes? Surely it could not be supposed that an honorable Commission would mislead for the purpose of concealing the fact that they had sent such unsuitable cases and so many of them? Nor could the thought be entertained that an honorable Commission could deliberately send cases known to be incurable organic cases, for the purpose of entrapping Dr. Stevens into their acceptance. In this connection the Commission has unfortunately placed upon record a statement which is to be regretted. In the report of the case of Stephen W., the Commission states: "The epilepsy was thought [by the Commission] to have an organic basis in a cicatrix in the left hemisphere; but, in spite of this statement, Dr. Stevens accepted the case."

The circumstances of the acceptance of this case were as follows:

The patient made his first call late in the day, and without any examination he was told to call at an appointed

time. The Commission was notified and both Dr. Webster and Dr. Moore were present for the examination. A large number of private patients were at the time demanding my attention. I requested Drs. Webster and Moore to make the ocular examination, which they kindly did. I did not go into the room where the ophthalmoscope was used, nor did I at that time even read the notes of the case that were brought by the patient. I assumed that the Commission would send none but suitable cases, and I doubt not that the gentlemen who examined the eyes and reported nothing noteworthy, made the examination in a much less careful manner than they would have done had they been expecting to find disease. Of course neither of us exercised sufficient care and on my part the neglect was an important one.

The case was accepted, and several days passed before I found time to make a critical examination. Soon after this I took the opportunity to express my belief to Dr. Dana that the case was an entirely unsuitable one, and it was withdrawn. I did not at that time suppose that it was the deliberate purpose of the members of the Commission to send a case which they had had under observation and which was known to be incurable.

1 The plan provided that typical cases only should be chosen, which were not complicated with any known organic disease and which had resisted approved methods of treatment. I assumed at that time that any case referred to me by the Commission I could accept in good faith. Experience, however, in this and other cases which were accepted at the beginning, led me at a later period to make careful scrutiny, with the result that quite a number of the cases sent were rejected as unsuitable. And this state of facts may throw light upon the reason for my insistence upon a full compliance with the agreement of April last, that the whole number promised should be sent, for of the early cases sent nearly every one should have been rejected, and four suitable cases would, at a later stage of the work, have been an important factor.

There is a familiar proverb that "Straws show which

way the wind blows." In ascertaining the spirit in which the report is made, it may be worth while to recall to mind the fact that in the history of one of the cases the report concludes: "In a letter of Dr. Stevens, dated November 7th, 1888, he (the patient) is alluded to as the "chronic boy."

The latent humor contained in the above sentence only becomes gradually manifest when we remember that a good deal of attention has been devoted to the person who is represented as characterizing his choreic subject as "the chronic boy." Dr. Stevens freely admits that his penmanship is not such as would obtain for him a position as writing master, but there is not this poor excuse for this attempt to subject to ridicule the man whom this Society has invited to all this labor and responsibility. The quotation which can have but one significance is not found in any letter written by me. I need not adduce further examples of the spirit which pervades this report, but I may call the attention of every one who has listened to the reading to the fact that whatever may have been the purpose or motive of the author of that paper, from its first page to its concluding clause, there is no word or sentiment which would lead the hearer to suppose that the person whose work has been so long under review is worthy of any confidence or of ordinary respect. What the clear tenor and effect of such a report is, I leave for the judgment of honorable men to decide.

Passing now to the reports of cases which have been under treatment, we are at once struck with the fact that so many of the cases which had from five to twenty years resisted the effects of toxic doses of arsenic and bromide were of such a simple and easy character. Again, it is worthy of note, that where two neurotic manifestations existed in the same patient, the disappearance of the one not specifically mentioned in the plan is made to account for the improvement in the other. Still further, cases which the Commission say have not been seen by the members for some time and concerning which cases they say that no conclusions can be arrived at, are reported among the "unimproved,"

while one of the cases which has been seen by the representative of the Commission, who has from time to time recorded the most favorable observations, is included in the class "unknown," the report stating that this is from a want of recollection.

It is my purpose here not to take up in detail every one of those reports, but to point out certain great defects in a few of them, which will throw some light upon the spirit in which all have been written.

Let us examine some of these cases.

First. The case of Elizabeth C. The Commission conceals that this patient is better, but not well, and that the first improvement appeared thirty weeks after beginning treatment.

The report fails, however, to set forth the heroic treatment to which this feeble little girl had been subjected during the two years prior to being sent to me. It does not state that she was, by the direction of the chairman of the Commission, kept month after month closely confined to her bed, nor that, according to the notes sent by him to me, she had taken under his direction twenty-five drops of Fowler's solution of arsenic, three times a day. It neglects to state that after these and many other heroic methods, the child was far more feeble when she came to me than she had ever been before, and that she was anæmic and destitute of every appearance of vigor or elasticity. It fails to set forth the facts that appear in the notes sent to me that her one or two attempts to attend school during the two years and more had proved absolute failures.

What are the facts concerning her improvement? During the school year commencing in September, 1888, and ending in July, 1889, the child attended school without the loss of a day, except when she was called out for Dr. Seguin's examinations, and during the present year she has renewed her school work with vigor and with no loss of time, except from the cause above mentioned. Her father, a professional nurse, and her mother a very intelligent woman, observed a marked improvement in her condition long before she was sent to school, and they are both confident that she was

able to have attended school much sooner than she did, but that their former experience led them to hesitate for a time, and then they concluded to wait for the beginning of another school year. The statement that the first improvement was thirty weeks after treatment commenced, is not in accordance with the facts as understood by the parents of the child, or as appeared from my own notes.

The report adds, "At present she has diplopia for near and far distance—an ocular defect acquired during treatment."

On reading this statement in the report I was surprised, for I had suspected nothing of the kind. On examining the child I found no evidence of diplopia; but I learned from her that what she called double images under the examination by the Commission, was the aberration which is perfectly familiar to oculists as the result of astigmatism. The child has three-fourths dioptré astigmatism in the left eye. Without a correction of this defect the over-conscientious child, when assured that what looked to her, as she expressed it, "Kind of double," really meant double, admitted that she saw double. In my examination, as soon as a three-fourths dioptré cylinder was placed properly before the eye, the aberration was corrected and the supposed diplopia promptly disappeared. I have so great confidence in the integrity and candor and the friendly sentiments of the oculists connected with the Commission, that I believe that if either of them is responsible for this statement, which is certainly an injurious one to me, he will improve the first opportunity to correct the important error. The child not only has no diplopia, but she has a condition of orthophoria with absolutely free and perfect rotation of the eyes in every direction, with abduction of 8° and adduction of 40° .

It is proper here to add that the Secretary of the Commission was kind enough to say that I could obtain the report of the oculists if I asked for it. As I have not yet sent a request, I have not seen it.

Second.—Flora K., Case 2. The report states that this case was under observation of the Commission four years previous to being referred to me, during which she averaged five attacks a year of grand mal.

The report fails to state that the patient had also great numbers of slight attacks in addition to the above, and that the patient had been an epileptic with severe and frequent fits since her first year (being at this time twenty years old). Nor does the report include the fact that at the beginning of the observation of this case optic nerve atrophy, with pathologically enfeebled action of all the muscles within the orbit, including drooping of lids, not amounting to complete ptosis, had been observed ; nor that attention had been repeatedly called to this condition. The report does, however, say that positive harm has been done by the ocular treatment, and proves it by setting against the five monthly attacks of grand mal and the unreported number of petit mal, the number of attacks of slight vertigo which, to use the words of the patient's mother, "Can hardly be noticed," and which from the time she first came to me she had been instructed to record at the moment on a calendar, which she is ordered to carry in her pocket for the purpose—a practice adopted only after she came to me.

The patient believes that she is greatly improved. The mother is sure that she is, and says that she can now trust the girl to go about alone, which she could not do before. She has almost completely recovered from some most distressing nervous symptoms which have from the beginning accompanied her disease. Her general health has greatly improved, and she is greatly better in her mental condition.

The patient has not recovered and does not belong to the class of functional cases. She long ago passed out of that class. She is certainly better, notwithstanding the remarkable statistics of the Commission.

Third.—George K., case 4. This was one of extravagant electric chorea with coprolalia. His clonic convulsive movements, his loud whoops and barkings, and his constant repetitions of obscene phrases under all circumstances are to his friends among their most painful memories, and to many physicians among their most curious observations. He has been widely known as the "barking boy." A month after ocular treatment commenced, a very remarkable change for the better was observed by all who saw

him. The mother says, "I thought he was well." Two months later a relapse occurred, but he did not return to his former condition; and both the boy and his mother positively assert that in his worst periods, since he has been under my charge, his condition has never been so bad as when at his best during some years before. The violent convulsive jerkings which were repeated every second or two have given way to slight movements often intermitting for a long time, and his loud barkings and whoopings have been replaced by a sound resembling a hiccough. Even these nervous manifestations are often absent altogether. He is not well, but notably better; indeed, judged by the ordinary standard of success in such cases, he is remarkably better. He is classified with the unimproved in the report.

Fourth.—Aggie H., case 5. This girl is said by the Commission to have had from two to four attacks of petit mal daily for four years.

The mother of the child has stated more than once in the presence of the members of Commission and others that her number of attacks very greatly exceeded this number, and that from forty to sixty would correctly represent them, that they occurred every few minutes, and that as no one could keep perpetual watch over the child her attacks could not be counted. Referring to her mutterings when in an attack, the mother says, "She was talking all the time." The mother now declares that the number of attacks is less than one-twentieth the former number, and that the character of them has been greatly modified for the better. Thus, notwithstanding the assertion of the report that "the result of ocular treatment pursued with regularity for fifteen months seems to be entirely negative," a really notable improvement has, in fact, taken place.

Fifth.—G. H., case 8. The Commission states the number of attacks of grand mal (which were attacks lasting several hours) at three in a year, another lighter form of epilepsy once in three or four weeks, and attacks of petit mal three or four daily. Thus it appears from the report of the Commission that the patient suffered from what, by the numerical method, which appears to be a favorite one of

the Commission, must have been many hundreds of attacks in the course of a year.

The Commission states in respect to the result: "Through lack of definite data no conclusion can be drawn from this case."

It is proper to inquire what data was and is in the hands of the Commission. It appears that of the four occasions when this patient has been brought to the city since he was referred to the Commission, he has been seen by one or more of the members on three. On each of these occasions written memoranda have been made. The following notes are in the handwriting of Dr. Birdsall:

NOTES MADE MAY 25, BY DR. BIRDSALL.

"Grandmother states that he has not had any convulsions, nor any of the attacks of vertigo (premonitory spells) since March 28th. Patient himself states, that about April 27th, he had, one day, the feeling that he was about to have an attack, resolved that he would think of something else; it did not come on, and none has occurred since. Tenotomy was first performed, April 6, 1888."

"March 30, 1889. Patient's general appearance is better than when last seen—looks as if he had increased in weight. Patient states from memory that he thinks he has not had any of the premonitory attacks, but he had one severe one this month."

This memorandum was made after a close examination of the patient, a very bright boy, and his father a man of learning and ability—an examination lasting more than half an hour. The claim of the report that nothing was learned regarding petit mal in all this long examination, and, therefore, that no conclusion can be formed, is remarkable. In fact, there were no such attacks. It will thus be seen that, according to the records made by one of the Commission, the patient had but a single attack in more than a year. Notwithstanding the fact that the Commission has a copy of these notes in its possession, the result of treatment is recorded as "unknown."

When we consider the professedly minute memory of the

Commission in certain respects, this lapse seems extraordinary.

Was this case relegated to the class "unknown"? because if it had been placed where it belongs it would have made the list of "improved," disproportionate to a number which would sustain the Commission's general conclusions?

Light may perhaps be thrown upon this question by a further interesting study of the methods of the Commission in presenting its professed summary of results, and which appears in the disposition which it has made of two cases, neither of which, according to the articles of agreement, should have been included in the report.

The Commission, for its own purposes, and contrary to the rules, established a time limit by which the case of David S. could be admitted to the list of those which the Commission calls "counted." Now, David was, as the Commission claims, under treatment four months; and stopping before his complex muscular errors were corrected, he did not get better. He is placed among those whom the Commission "counts" and in the list of "unimproved."

Patrick H. was another epileptic who dropped out, but Patrick was much longer under treatment than David. If the rule included David among the "counted," why not Patrick? There is this coincidence—Patrick, when submitted by the Commission, had an average of more than two-fits of *gros mal* every day—fits in which he fell in convulsions. A remarkable falling off of these attacks was observed during the first month of his treatment, for during that month he had only seven such attacks. The following month there were four; the next month one; then there was an increase to four during the fourth, and again a decline to two in the fifth month, and during the sixth month he did not have any. Patrick thought he was pretty well and discontinued attendance. Now, if the case of David, who did not during his four months get better, was to be used to swell the list of the "unimproved," by what logic was Patrick, who, during his six months of attendance, had shown remarkable improvement, placed in the list of "not

counted" and thus prevented from serving a similar purpose for the column of "improved" which the other had served in the opposing list?

Sixth. The last of these case reports to which I shall call attention is Case 13, Mary McK. This patient is a woman of excellent intelligence and who, notwithstanding her malady, which has kept her much of the time away from her duties, has been continued as a teacher in one of the Public schools. She was examined by members of the Commission in March, 1889, and in reply to their question gave an approximate estimate of the number of her convulsions. Before being sent to my office about the first of May last, she was handed a calendar on which she was instructed to record with greatest precision every convulsion and every dizzy spell of whatever degree of severity. Since she commenced treatment directed to ocular conditions, six months ago, she has greatly improved, although the bromides have been discontinued. She had, about the middle of August, an attack such as she had reported to the Commissioners as of frequent occurrence. She has had none since. She has in the meantime experienced some momentary sensations of vertigo, which have seized her while standing in front of her class, but have caused neither staggering nor falling—nothing more than a momentary sensation. These little attacks she at once records and these, which were not included in her statement to the Commission, are counted against attacks of convulsions in which the patient fell and was unconscious. It may be that this kind of numerical method of estimating the degree of epilepsy is a correct one. But in spite of the verdict of the Commission that she is unimproved, the patient and her friends think that there is a marked difference between falling in a fit from two to four times a month and having a dizzy spell as often. Especially do they think this since they assert that, in addition to the fits reported originally, the dizzy spells were even more frequent and severe than at present. This patient and the friend with whom she resides declare in the most positive manner, and in the presence of many gentlemen eminently competent to judge of the value of the statement,

that she is incomparably better. And the contrasts to which I have drawn attention, between the actual results and the results as set forth in this report in the cases which I have before cited, can and will be in every instance confirmed by these distinguished gentlemen.¹

It is unnecessary to pursue this subject further. It has been shown that the ordinary standards of judgment have been absolutely disregarded in these cases. It is easy to observe in the report of every case a sort of unilateral method of presenting the subject, suggestive of a kind of mental hemianopia, which certainly makes it appear greatly to my disadvantage.

The various facts which I have set forth may aid us in interpreting the comparative degree of success or failure of the work.

An inquiry which forces itself upon every mind is, what good was to be secured by prolonging this investigation through all this time by the failure of the Commission to send the stipulated cases. We have already seen that at the end of the first year, excluding three that were unfit, or did not come, the Commission had furnished, in all, but six cases. It must be admitted that this Commission either chose not to send cases, or it could not find them. If it could not, after a year of trial, furnish more than half the cases required by the agreement, should the Commission not have abandoned the trial or surrendered it to those who were possessed of larger opportunities?

Is it the judgment of this Society that the Commission had satisfied the conditions of the challenge for which the Society became responsible?

When the investigation was proposed, I assumed that all these cases would be forthcoming at once. I could not have supposed otherwise. The whole tenor of the agreement shows that that was the understanding. Is it to be believed that one would have consented to such an investigation had he supposed that five professed neurologists would require a year in which to select six cases of sufficient difficulty for the investigation?

¹ See letters from these gentlemen appended to this reply.

The Commission claims that it did its best. We are at liberty to inquire what was its understanding of the best methods of selecting its cases, and the following example will serve to illustrate what methods it actually adopted:

September 28, 1888, L. G. called with a letter from one of the Commission requesting me to examine her with reference to her acceptance as a Commission case. She was an epileptic, but to my great satisfaction free from any discoverable organic lesions. She was in good general health; had rosy cheeks, was not stupid from bromides, and was, for a Commission case, a decided novelty. One eye was myopic, the other astigmatic, and she had 8° vertical diplopia. In my satisfaction at getting a good case, I wrote, perhaps a little enthusiastically, that she was a good case and that she had vertical strabismus.

The case did not return, and I soon learned that one of the oculists of the Commission had operated for her strabismus. On calling the attention of the Commissioner who sent her, he replied: "Oh, yes, she was found to have vertical strabismus, so was sent to Dr. —, who has operated twice."²

Is there a member of the medical profession who would be unable to imagine that, with the above method of furnishing cases, a year might suffice to obtain some half-dozen which would present more than ordinary difficulties? Whatever may have been the original condition of the majority of the cases sent to me during the first year, it is certain that they had at that time long ceased to be cases of functional disease.

It is not to be forgotten that all the cases upon which the Commission claims to base its conclusion, and which were sent by the Commission, had become worse under all forms of treatment. The testimony of the Commission is conclusive on this point. For example: David S., who had taken bromides indefinitely and in large quantities, stopped

² This statement was confirmed during the discussion by the member of the Commission who sent the case.

the medicine April 23d. The report states that "the result of stopping the bromides in April was the occurrence of nine severe fits between May 6th and 9th."

Again, John C., who had epilepsy twelve years and was saturated with bromides, had according to this report, thirty-one severe convulsions during the month succeeding the discontinuance of the drug. Dr. Seguin became so greatly alarmed lest the patient should die, that he restored him to the benefits of the bromide treatment which, if the patient yet lives, he is still probably pursuing.

The report states that "In all these cases (of epilepsy) the immediate effect of withdrawing the bromides was to cause an increase in the number, and in some cases, in the severity of the attacks. This rarely continued longer than three months." In other words, after three months of ocular treatment, improvement occurred, and this improvement has not only continued, but the patients who continued under observation, without exception, have made steady improvement.

Thus, while as a matter of fact every one of these unfortunate cases belonged to the class of incurables, by any of the ordinary methods of treatment, and as a matter of record—shown by the Commission—these patients had not only not improved, but had grown worse under such treatment, continued during many years by themselves or others, whose treatment they have approved, the cases properly included in this report, have in every instance under the present mode of treatment, materially improved in health and in respect to the special disease for which they were treated.

Indeed, in several instances very notable improvement has occurred. All this has been brought about without recourse to the deadening results of bromides or the toxic effects of enormous doses of arsenic.

In concluding this review of the report of the Commission, permit me to recall the salient facts in the case :

From the beginning the Commission entered upon its work in a manner entirely inconsistent with the true spirit of inquiry.

It pursued it in a manner calculated to induce vexation, misunderstanding and controversy.

It occupied many months in securing a few cases and has never yet complied with its own stipulations.

It has, through more than two years, occupied itself in selecting the most unfavorable cases for treatment, and has taken the favorable case to itself.

The spirit of the report is calculated to produce prejudice and tends to mislead.

The report makes professed quotations which are calculated to subject Dr. Stevens to condemnation or to ridicule, and the letters from which these professed quotations are said to have been made, never, in fact, had existence.

The report is now made in violation of every principle of right or fairness.

The report is represented as an investigation of two diseases and not of a principle, in direct violation of the pledges of the Commission.

The Commission has repeatedly sent cases well known to be unsuitable for the inquiry and has, contrary to the articles of agreement and contrary to the private promise of members, in this report paraded these cases in long detail, with the effect of misleading the judgment of those who are unacquainted with the facts.

It has reported the results of treatment of the cases by minimizing the importance of the malady at the beginning of treatment and by magnifying all the unfavorable conditions now existing.

The results represented are not in conformity with the actual facts, but are grossly at variance with them.

It has reported as resulting from treatment, unfavorable ocular and nervous conditions which do not exist, and has, in this respect, made statements which are grossly at variance with the facts.

NOTE TO DR. STEVENS' REPLY.—On November 4th a number of gentlemen, all well known to the medical profession, examined eight of these cases, including all that had been under treatment during the past year, and who reside in New York or its vicinity. Those not included reside at distances varying from two hundred to one thousand miles from the city and could not be present.

New York, Nov. 5th, 1889.

My Dear Doctor:

The facts that I have elicited from the patients shown last evening to me at your office by my personal inquiries are these. As to

1. Elizabeth C., aged 16, presented as a case of chorea, the mother states that the patient is now quite well, except under much excitement when some mild indication of the former trouble shows itself. The child was free from any signs of chorea at the examination.

2. George K., 14, chorea. Known as the "barking boy." He had been seen by me several times in the past two years, when he barked and jerked very badly. Last evening when exhibited, his motion and noise had very much diminished, and the patient and his mother state that he is greatly improved, and so he seemed to me also.

3. Miss McK., epilepsy. The patient stated that she has never been better in her life than now. That she had not had an epileptic seizure since August 19th, of this year. That prior to treatment she had had as many seizures as five or six a month. That her spells of "confusion" have also much lessened in frequency and duration. These latter had not been counted in her previous reports and were considered by her as distinct from her decided convulsive attacks.

4. Gertrude W., epilepsy. The mother stated that the patient had had four fits prior to Dr. Stevens' treatment and three fits since. That the smaller attacks had diminished from five or six a week to once or twice a week. She considered her daughter to have been much improved.

5. Flora K., young woman, epilepsy. Her mother said the patient was very much better than two years ago, that the attacks were less frequent and less severe, and that the improvement had been greatest in her condition within the past two months. The patient herself stated that she was much improved.

6. Eva S., young girl with chorea. Not much change in the patient's condition the sister reported, though the duration of the quiet times had increased.

7. Agnes H., a young girl with epilepsy. Under treatment since last April. Up to nine days ago no change in her condition, she up to this time having had two or three attacks a day (similar in number to the period prior to April). For the past nine days no attack whatever.

8. Aggie H., 12, with petit mal. Now having some days one attack, some days none, the mother states. She further said she never expected her child to be as well as she is now. That she is more than twenty times as well as she used to be.

In the examination of the condition of these patients, I may add that no details of the treatment were asked for or considered by me.

(Signed), R. F. WIER.

To Dr. George T. Stevens.

I have read Dr. Weir's report, and here state that Dr. Wier's statement agrees with my own in every essential particular.

(Signed), GRAEME M. HAMMOND.

19 West 21st Street, Dec. 7th, 1889.

DR. GEORGE T. STEVENS.

Dear Sir—I had the pleasure of examining at your office, on the evening of November 4th, 1889, eight cases of chronic epilepsy and chronic chorea. Elizabeth C., George K., Miss McK., Gertrude W., Flora K., Eva S., Agnes H. and Aggie H.; cases referred to you by the Commission of the New York Neurological Society, and who had been subjected to your ocular treatment at their request.

The histories of the patients, as presented to you by the Commission, were read, and then we were allowed to question patients and their friends, and to examine the patients as much as we chose. Being guided wholly by the antecedent history as to their condition when you took charge of them, a careful examination of each case convinced me that *all* had experienced benefit from your treatment, which benefit seems greater when we consider that at the time of your assuming control *all medication* was stopped.

While *all* seemed improved, and testified to great improvement, it seemed to me that the patients Aggie H., George K., Elizabeth C. and Miss McK., in the order named, showed most improvement.

Very truly yours,

(Signed), R. W. AMIDON.

Dec. 7th, 1889.

DR. GEORGE T. STEVENS.

My Dear Sir—On November 4th, 1889, I had the opportunity of examining at your office, in conjunction with several prominent medical gentlemen of New York City, eight cases of chronic epilepsy and chorea; all of which had been subjected to ocular treatment exclusively at your hands in connection with the work of the "Stevens' Commission."

At the meeting every opportunity was afforded each of the medical gentlemen present by yourself to examine these patients without restraint, and to obtain for themselves a full knowledge of the facts without aid or suggestion from you.

Many of the patients shown at that meeting had been seen by me in your office from time to time while their treatment was being conducted by yourself; hence I was quite thoroughly familiar, from personal knowledge, with their physical condition before ocular defects were either combatted or fully corrected.

From memoranda taken at that time, I take pleasure in stating that, in my judgment, no case failed to show marked improvement. In several cases the results were to me quite startling, as I had not seen them in several months.

In the case of George K., the so-called "barking boy," who, as far as I know, belonged to a type of sufferers that is generally regarded by eminent neurologists as incurable by any known therapeutic measures, the improvement was very remarkable. His barks were only a slight "hiccough" and infrequent. His head was not forced between his knees, as it was every three seconds when I first saw him, and his mother's story of gratitude for the improvement in this respect, as well as for the quiet sleep which he has enjoyed since being under your charge for the first since his disease commenced, was evidently heartfelt.

Among the others in which I observed the greatest improvement, I would mention the epileptic Miss McK., the epileptic girl, Aggie H., and the choreic girl, Elizabeth C.

In the case of Aggie H., the mother states "the child had from forty to sixty attacks of petit mal daily when the eye treatment was commenced, and that now she has sometimes one and often none at all." She also said that the attacks which she does have are now lighter than they have been in the past, and that the child is better in her health than ever before since epilepsy developed.

Respectfully yours,

A. L. RANNEY.

The full histories of the cases will appear in the December number.

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Meetings of November 5th and December 3d, 1889.

DR. GEORGE W. JACOBY, President, in the chair.

The President called for the report of the Stevens Commission. Prior to the reading of the report, the following protest was presented by Dr. G. T. Stevens :

“ Mr. President and Gentlemen of the Neurological Society :

“ I respectfully protest against the reception of the report now announced, for the reasons—1st. That the Commission having failed to perform its part of the agreement upon which the report is to be founded, the report is not now in order. 2d. Because the report is not a report on the proposition at issue. 3d. Because the report is inconsistent with the facts, and is misleading. And 4th. Because it is an ex-parte report, and objectionable in spirit and motive.”

The report of the Commission was then called for by the President, and read by its chairman, Dr. E. C. Seguin (see page 657).

Dr. HAMMOND had seen eight of the cases described, in company with Drs. Weir, Webster, Ranney, and Amidon, the day before the meeting. The patients and their friends all admitted that the cases had been improved very much by Dr. Stevens' treatment, a fact which was at great variance with the statements made by the same patients to the Commission.

Dr. SEGUIN said that that part of Dr. Stevens' remarks which reflected upon the fairness and integrity of the Commission needed no reply, the names of the members of the Commission being a sufficient answer. Dr. Stevens' statements as to the evil effects of the arsenical treatment of chorea, and of the bromides in epilepsy, appeared to pander to popular prejudice, and were altogether unwarranted by facts. Some clinical experience and knowledge of practical therapeutics would have prevented such expressions. Arsenic was almost never harmful to choreic patients, and the evils of bromism could be prevented and corrected by watchful management and appropriate associated treatment.

It was true that the Commission had had the greatest difficulty in inducing patients to undergo the treatment, but their failure to obtain twelve cases within the first few weeks was not through neglect or violation of the agreement. It was simply an impossibility to supply the necessary material in a given time: enough had been furnished in all. Dr. Stevens, in not furnishing data as to the course and progress of the cases as stipulated in the agreement, had violated the compact. The report had no personal animus, and was not made up of the ideas of any one or two members. It expressed the

opinions of all of the members of the Commission, even of those selected by Dr. Stevens himself.

As regarded the statements of friends and relatives in medical investigations, these should be received with scientific doubt, particularly when they differed from the facts, as shown by the Commission.

As far as his own cases were concerned, one had been nearly killed by the ocular treatment involving the omission of bromides, passing into a status epilepticus, but improved again under bromides. Another had been under ocular treatment for fifty-two weeks before improvement became marked. Another, the girl with chorea, showed no tangible improvement until after the lapse of seven months. His own experience in cases of chronic chorea, even when of five to seven years' duration, was that they were not merely improved very soon under arsenical treatment with rest in bed, but, as a rule, cured in from two to three months. This girl, though improved under the ocular treatment, had now transient diplopia for far and near, while the second case alluded to saw double at the present time on looking to the right. The improvement in these cases (which were the best of the series) could not therefore be ascribed to the ocular treatment, but was to be attributed to improved health and time. Dr. Stevens had reported fifty per cent. of *cures* of epilepsy in his own (former) series of cases, but in the cases examined by the Commission there was not one. Moreover, he had reported his cures as taking place in a few weeks after beginning the ocular treatment, while in these cases where improvement of any kind had taken place, many months had been required before decided change had been manifested.

Dr. STARR, in regard to the remarks made by Mr. Hammond, referred to the records of the cases read to the Society, which demonstrated in each case an increase in the number of attacks, in some of them to three times as many as formerly. It was well known that the statements of epileptics were unreliable concerning their own seizures, and in several of these cases it was evident from their own records of their attacks that any statements that they had made that they were better were in direct opposition to the real facts. The recollections of such patients regarding their past states could not be considered as at all comparable with the actual observations taken during such periods.

In one case, pronounced by Dr. Webster as normal ophthalmologically, and in every respect a purely functional case, Dr. Stevens claimed to have found optic atrophy and called it an organic disease.

Dr. STEVENS said, referring to the lad, George K., who has been introduced here by the Commission and made to face this audience for an hour, this case was included not in the list of improved, but of the unimproved. On the blackboard had been written the number 5 as that of the improved. Since the lad by his quiet demeanor and eloquent silence had shown his true position, the Commission admitted his improvement for the first time. This is only one of

several concerning which such a change should have been made. This report includes two lines of statement. The first relates to the dealings of the Commission with himself; the second relates to the professed results of the work.

In respect to the second line of statements, it was difficult to place before the Society the real facts. He had therefore requested a number of gentlemen, distinguished for their fairness as well as for their great ability, to see several of the patients, who have been declared by this report unimproved by treatment. These gentlemen, after a careful examination, had arrived at conclusions widely different from those represented in the report.

If this line of statement could not well be presented, that relating to the dealings of the Commission could better be shown, and by the manner in which the Commission has treated this part of the report, the whole report should be judged. The length of time occupied in selecting cases indicated the kind to be eliminated and the kind which were to be sent. Dr. Stevens inquired whether the Society would indorse the professed quotations and the incorrect statements with which the report abounded, and which were without meaning or consequence, except as they might bring into discredit and ridicule the man invited by the Society to do this work.

Unquestionably many of the cases sent by the Commission and accepted in the beginning of the inquiry ought never to have been taken. Few of these early cases were at that time any longer functional.

The operative technique in many of these cases had proved exceedingly delicate and difficult, and had demanded a longer time in their management than ordinary cases. The greatest patience and skill had been required, and he did not profess to have attained the skill demanded for the quick correction of anomalies such as were here found.

He had not, as the Commission had stated, asked for favors or cases, and he had never written letters which are attributed to him by the Commission. Dr. Bird all had himself answered one of these statements when he said in this discussion, "Dr. Stevens did not ask for four cases. It was the proposition of the Commission."

Dr. BIRDSALL had seen an epileptic, one of the cases mentioned by Dr. Hammond as having reported improvement to him, and whose sister corroborated the patient's statement. He had asked the sister why she had made this statement. She had replied that she did not refer to the number of fits, which was unchanged, but meant that the patient seemed to be brighter without the bromides. The record, showed, however, that the number of attacks had increased and that the patient was in reality much worse under Dr. Stevens' treatment.

Dr. DANA said he had been appointed on the Commission by Dr. Stevens himself and felt that he represented him in a measure, but naturally his chief object had been to obtain the exact truths in the

matter. He had sent twelve cases of his own to Dr. Stevens, and had tried hard to get others to go; but he had seen no case much improved under Dr. Stevens' treatment. One case seemed to be improved, but he could not feel sure that improvement was due to the ocular treatment. He had lost most of his faith in it as a therapeutic measure. We ought, however, pay some tribute to Dr. Stevens for perfecting the technique in his procedures, even though he had pushed his theory to extravagant conclusions.

Dr. WEBSTER stated that the diplopia now existing in one of Dr. Seguin's cases did not amount to much. It was trivial. The patient had stated to him that it produced no annoyance. He had seen Dr. Stevens operate many times, and had learned very much from him as to the technique in the treatment of insufficiencies. Dr. Stevens had, moreover, greatly improved the nomenclature in this direction. Not being a neurologist, he did not feel competent to judge of the therapeutic effects of the operations in these cases.

Dr. L. C. GRAY had listened with interest to the report and its discussion, and was wholly unprejudiced on either side. He did not believe that Dr. Stevens had substantiated his serious charges against the Commission, or caused any particular improvement in the cases. Esquirol showed in 1828 that temporary improvement took place in all cases of chronic epilepsy in La Salpetriere when treated by drugs known at that day. A decrease of from six to three fits daily could not be considered a permanent improvement, because such decrease was often spontaneous, and in some cases fits spontaneously ceased for years. He agreed with Dr. Seguin that where improvement did not begin before fifty-two weeks after treatment had been begun, any improvement that might take place could not fairly be ascribed to the measure employed. If Dr. Stevens had claimed merely that headaches and other trivial neuroses had been relieved by his method, many of the gentlemen present would have coincided with him, but he had undertaken too much and was extreme in his claims. The same therapeutical principle underlay his system as that of Sayre, who cut prepuces, and that of the English surgeon who amputated clitorides for the cure of the slighter neuroses.

Dr. AMIDON said that he had been invited with a number of others by Dr. Ranney to see the cases described at Dr. Stevens' office, and they had the complete histories of the cases at the time upon which to form an opinion. He intended to oppose the acceptance of the report of the Commission, for it was clearly apparent that Dr. Stevens had brought about improvement in these patients without resort to medication, which was more than any of us could do.

Dr. SEGUIN said, with reference to the work done by Dr. Stevens, that all of the member of the Commission had appreciated it highly and had felt like adding something commendatory of his skill to the report, but refrained from so doing because, under the circumstances, it might have appeared hypocritical, and was really alien to the subject assigned to the Commission, viz., the determination of results.

Dr. J. L. CORNING thought that Dr. Dana's tribute to the good work done by Dr. Stevens was in exceedingly good taste. Dr. Stevens' work was unusually scientific and original, and there seemed to be very little of that sort of work done in New York, where compilation and the importation of foreign ideas were the order of the day. Hence this discovery of Dr. Stevens ought to meet with wide recognition.

A motion to adopt the report of the Commission was then carried by a vote of five to four out of the eighteen members present, the members of the Commission refraining from casting their votes.

The following motion made by Dr. Amidon was unanimously carried:

"That the thanks of the Society be extended to Dr. Stevens for the assiduous labor he has devoted to the work of the Commission, and that the Society assure Dr. Stevens that it highly appreciates the ingenuity and technical skill he has shown in the prosecution of his method."

Besides the report of the Stevens' Commission, the following contributions were made at the two meetings of the Society:

Dr. IRAN VAN GIESON read a paper entitled, "A Contribution to the Pathology of the Laryngeal and other Crises in Tabes."¹

Dr. A. SCHAPRINGER presented a case of "Congenital Bilateral Abducens Paralysis with Facial Paralysis."¹

Dr. C. A. HERTER read the clinical history and report of the autopsy in a "Case of Cervical Paraplegia from Dislocation."¹

¹ Paper will be published in a future number.—Ed.

Orders for December number must be sent in immediately.

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(Concluded.)

THE FULL HISTORIES OF THE FOURTEEN CASES UPON
WHICH THE REPORT WAS BASED.

(See Report, page 657)

Each history is made up of the notes sent to the Secretary by the various members of the Commission on the various dates given at the head of each note. The summary was prepared by the member sending the case.

M. ALLEN STARR,
Secretary.

No. 1.—Elizabeth C., 13½ years old, chorea, Dr. Seguin.—First seen February 15, 1886. Parents healthy, free from neurosis. *Early history*—Normal. Child walked and talked at usual age. At nine years of age moderately severe attack of scarlatina, not followed by dropsy; never articular rheumatism. Did well at school until her thirteenth year when at Grammar School, in which she was put back one class. Patient worried very much over this, and studied at home. Always over-anxious about lessons. All this occurred in winter of 1884-5, preceding the *first attack of chorea*, which ran its course in May and June, 1885. The choreic movements were limited to the right side, but speech was unaffected and there was no paralysis. Under ten-drop doses of Fowler's solution the attack subsided. Was well all summer. *Second (present) attack.* Patient sent to school at opening of term (middle of September, 1885), and in two or three weeks the chorea reappeared in face and tongue first, then generalized. Tried school again occasionally afterward. On examination no symptom of organic disease of the nervous system exists. *Causes.* Masturbation was suspected and watched for carefully by the mother, who says she never detected it. There is no cardiac murmur. The apparent cause is cerebral strain by study and worry. *Treatment.* A thorough rest-treatment (in bed) with Fowler's solution to toxic effects, was tried in February and March, 1886, with only moderate improvement. The dose of twenty-five drops of Fowler's sol. three times a day was attained. Various tonics, the bromides, cold sponging, etc., were subsequently tried with only a moderating effect on

the chorea. Recently treated at the College of Physicians and Surgeons by Dr. H. W. Berg with Fowler's sol. of fifteen, eighteen, twenty drops three times a day. *Result.* Decided improvement. Patient cannot take more than eighteen drops without disturbance of stomach. *Mental condition.* Not hysterical and but slightly enfeebled. *Present condition.* April 17th, 1887. Mild generalized chorea. Speech slightly indistinct; rather childish; general health very good; no anæsthesia or paralysis; pupils normal; heart normal; patellar reflex high; no sign of menstruation; chest flat. Medicine stopped to-day. No conclusive evidence of masturbation, though there is some leucorrhœa.

(Signed), E. C. SEGUIN.
M. A. STARR.

April 17, 1887, is transferred to Dr. Stevens as a Commission patient.

NOTES OF THE CASE WHILE UNDER DR. STEVENS' TREATMENT

The memoranda furnished by Dr. Stevens and by the oculists of the Commission are very scanty. I have seen the patient at intervals of two or three weeks during the two years, excepting the summer months while away from New York, and carefully noted her condition.

1887. Dr. Webster's note, April 22d. "Miss E. C. examined under atropine at our office, examination without atropine by Dr. Stevens the day before. Esophoria 4°, abduction 4°, adduction 9°, no hyperopia.

$$\begin{array}{rcccl} & 20 & & 20 & \\ \text{R. v.} = & \frac{20}{50} + & : & \frac{20}{20} & \text{w.} + \frac{1}{42} \\ & 50 & & 20 & \\ & 20 & & 20 & \\ \text{L. v.} = & \frac{20}{50} + & : & \frac{20}{20} & \text{w.} + \frac{1}{42} \\ & 50 & & 20 & \end{array}$$

"Ophthalmoscopically normal."

May 10. Dr. Stevens writes Dr. Starr as follows: "A formula for glasses for Elizabeth C. has been given to-day, and her treatment may be considered as commenced. It is possible that it may be proper to make an operation for left hyperphoria on Friday next, at the time appointed for other operations. . . ."

May 18. (Dr. Seguin's note): "To-day has about as much chorea as when referred to Dr. Stevens. Mother states that she has at times been almost, but never perfectly free from chorea. The first menses appeared without pain on the 4th instant and continued seven days (patient will be fifteen years old in July). This event had no effect on choreic state; she simply looked a little pale."

June 1. "Chorea about the same as regards movements of the limbs. Mother reports that lingual and buccal choreic movements are worse than when medicinal treatment was stopped, two months ago; the speech is thicker, and in the performance of various voluntary acts the tongue is projected from the mouth, and smacking movements are frequent. Often drops objects. Menses was normal on twenty-eighth day, viz.: May 29. At close of conversation the chorea is undoubtedly worse than four weeks ago." [This was three weeks after glasses were ordered.]

June 15. "No change. Variable amount of chorea from day to day. Now does not jerk as much as when last seen. More chorea on left side."

June 25. By Dr. Stevens to Dr. Starr: "We will operate on Elizabeth C. (Dr. Seguin's patient) for hyperphoria, Monday, June 27th, at 1.30 p. m."

July 4. "No change in chorea. It appears that two or three operations were done last week: the first on June 27th. Father states that patient was quiet just before going to the operation, and *very* quiet after it. Was remarkably free from chorea for two or three days. Since then it has returned, and is now as at last note. General condition fair"

July 29. "Very slight chorea; jerks small and frequent, occurring in various muscular groups on both sides of the body. Tongue very jerky; facial muscles quiet. Has resumed glasses (spectacles) in last ten days, by order of Dr. Stevens' assistant (Dr. Boyer). Mother says she notices very little change in the disease."

Aug. 29. "Is to-day fully as choreic as at any time since beginning treatment. Variable degree of chorea on different days. Parents think that she is worse on the whole. Is pale and thin. Dr. Boyer suspended use of glasses seven days ago and ordered pil. ferri et quin." [It will be observed that nearly fifteen weeks after Dr. Stevens declared the treatment commenced the patient was worse on the whole, and that Dr. Boyer felt obliged to order a tonic, in violation of agreement.]

September 28. "Is undoubtedly better than at previous visit. Still has distinct choreic movements on both sides of the body, more on the left. Speech almost normal. Father states that this improvement began on the 23d inst., after beginning the use of last spectacles on 20th." [From other notes it appears that Dr. Stevens ordered two sets of glasses after his return in September].

October 4. Since last week (Wednesday and Thursday) renewed chorea of tongue, hands and legs. This aggravation continues. Patient is about as she was last summer. New glass over right eye on September 29th."

October 21. "Muscular twitchings in face, mouth, arms and shoulders very marked. Speech slightly affected. Tongue is often projected, and patient makes smacking or sucking noises. Mother states positively that as compared with patient's condition last spring, there is no material change." [This is twenty-one weeks after beginning of treatment. I urged the mother to submit the child to one more operation].

November 4. Note by Dr. Webster: "Miss C. having an exophoria of 2° + with an abduction of 11° , Dr. Stevens advanced the right internal rectus, which had been set back a little too far by its last tenotomy, and after the completion of the operation there was esophoria 2° , with an abducting power of 3° ."

November 21. Note by Dr. Stevens to Dr. Starr: "Dr. Stevens will operate on Eliz. C. (choreic patient of Dr. Seguin) for exophoria on Friday, November 25th, at 1.30 p. m."

December 9. Rather more chorea of hands; much more of tongue; while working or playing she "works" her tongue in her mouth constantly, and drools so that chin is sore toward evening. In reading articulates fairly well. Occasional jerks of legs. Another operation will probably be done to-day."

December 30. "Almost perfectly free from chorea. The least twitching

(the least possible twitching meant) occurs in fingers and about mouth. Speech normal. I have never seen her nearly so well. Father states that a week ago she was 'bad,' as bad as ever. Improvement began on the 24th and 25th; a rapid, but not a sudden improvement. Last menses on November 20th; none in December, and the aggravation of last week was at a time corresponding to menstrual period."

1888. January 11. "Menses last week: normal, with little pain; was not more nervous then. The great improvement noted at last visit continued three or four days. Since has had slight twitching in various parts: in face, neck and arms. In walking the left leg is lazy and weak. Has spectacles since December 9."

February 3. Remarkably free from chorea while sitting or simply standing. When talking, however, choreic movements appear slightly everywhere; most marked in right hand. Mother is still afraid to trust patient with dishes, etc., as she would drop them. Yesterday was not as quiet. Speech slow, but distinct. General condition good."

February 24. Memorandum by Dr. Webster: "Name, E. C. Physician, Dr. Seguin. Hy. R. 1°. Tenotomy of superior rectus. Aiter no Hy. Exoph. (? written 'Eoph.') 1°. Abd. 5°."

February 27. "Still shows the same slight universal chorea as when last seen; most marked in right hand and about mouth. An operation was done on the 24th instant in presence of Dr. Birdsall. Menses normal, and patient is not more nervous then. General health good."

April 4. "Is almost perfectly free from choreic movements, strictly speaking. While standing, and more while walking, has partial flexion movements of fingers at metatarso-phalangeal articulations (approach to cone-hand), and at wrist. The thumb is pressed hard against the index. This is a tonic movement. Mother states that when patient's hand is upon her arm in walking, she feels the fingers contract and 'draw.' Had more choreic movements after last operation (February 24th.) No facial movements seen during this visit. Has one patched glass in spectacles (over left eye)."

April 30. "Left eye was kept covered for three weeks. In last two weeks the right eye has been covered (with an opaque glass) and is so still. Is rather anæmic, though general condition is fairly good. Comparatively little chorea; more of right hand. Variations from day to day, e. g., speech was affected on 27th, but to-day is easy. Pleasurable excitement develops chorea more than annoyance or grief."

May 28. "Still exhibits traces of chorea in speech, right hand and leg. Speech affected only occasionally under pleasurable excitement. Right extremities show a little *slow* chorea all the time. Tongue a little unsteady. The patch was removed about three weeks ago, and she has since used both glasses."

June 6. Memorandum of operation (by Dr. Moore?): "Eliz. C. Exophoria 2°. Abduction 6°. Took a stitch in left externus, leaving after exophoria a trace, abduction 9°."

June 20. Remarkably quiet in last few days. Some impediment in speech, and occasional jerks of hands and face. Another advancement is to be done by Dr. Stevens on 22d. Is wearing spectacles, with both glasses clear. General condition good."

October 8. "No operation since June. Has grown, and stands better. Glasses have been changed twice in summer. Has very little chorea, but it is still distinct in right upper extremity from shoulder to fingers, and in right leg (flexion movements at knee). No facial chorea visible, but patient says that her tongue still moves involuntarily in mouth. Speech low, a little hesitating, but very distinct. Father states that there was more choreic disturbance last week. She has at no time been free from it (chorea), though there is certainly a great improvement."

November 16. Almost no chorea. A little still shown in right hand and arm. While standing she holds her right hand (by her side) in a peculiar position: thumb in, and whole hand cone-like. Had more chorea a week ago. Glasses were again changed with apparent benefit. Menses regular and free. General good health.

December 3. "Drs. Stevens and Boyer discovered, a couple of weeks ago, that she had been using a wrong glass! Changed. She had four or five different pairs of spectacles. Left externus cut last week. Much more chorea in last three weeks; tongue a little unmanageable, right hand and arm and leg (little) quite active. More chorea than for a long time."

1889. January 23. "Hands about the same; speech remarkably good. On 19th inst. right externus was cut. No glasses since. Father states that, on the whole, there has been no change in three months: a little better or worse at times."

February 22. "Almost no chorea. A few twitches in both hands, and some pretty constant motion of right fingers. Speech very good. Improvement followed the last operation in two or three days"

March 22. Another operation was done about one month ago. A week later, the patient felt something slip or give way in the right eye, as if it were "strained." Two weeks ago, prism-glasses were ordered for reading only. Is without glasses now. At no time diplopia; while sitting is almost perfectly quiet; the small twitches occurring, now and then, in the fingers while standing; however, distinct choreic jerks occur in both hands and knees—more marked in right side; the patient and her father are both cognizant of this. Pulse and tongue normal; general condition good.

October 21. Presents no choreic movements. Speech good. Mother states that, occasionally during the last three months, has had slight thickness of speech and jerking of the hands, usually before the menses—which have been regular and easy. Patient looks well. Can read from one half to one hour without headache. Is troubled with diplopia in reading, not at a distance, but by placing red glass over one eye, heteronymous diplopia at twenty feet is demonstrated. With the qualification that there are occasional slight recurrences of chorea, this patient may be considered as cured. She has, however, in the course of treatment acquired an ocular defect, viz.: diplopia, both for near and far distance. Since May, an operation on the left rectus internus has been done, and two sets of glasses given. She wears her glasses only for work.

October 29. I find in Eliz. Cr.:

$$\begin{array}{rcl} \text{R. v.} & & 20 \\ & = & - \\ \text{L. v.} & & 15 \end{array} : \text{Hm. } 0.25 \text{ D.}$$

Hyperphoria 0° , Exophoria, 1° , in accom. 4° ; sursumduction R $\frac{3}{4}^{\circ}$, L $\frac{3}{4}^{\circ}$, abduc. 5° , Adduc. 16° . Movements of eyes do not seem limited in any direction. No diplopia on testing with red glass. Ophthal normal. Patient says she has had thirteen operations; that when she looks up from reading, suddenly, she sometimes sees double, and that her eyes smart and burn, and run water when she reads. Her mother says she does not complain of it very often. There have been very few manifestations of chorea since last May.

(D. WEBSTER.)

October 30. Examination reveals the fact that diplopia is variable and occasional. No diplopia with red glass to-day.

(SEGUIN.)

Summary.—A careful perusal of this tedious journal reveals on the whole a great improvement—almost a cure. It will be noticed that the “treatment,” i. e., the use of spectacles and the performance of several operations, was not quickly followed by improvement, as it appears that on October 21st, 1887, twenty-one weeks after the official “beginning of treatment,” the chorea was as it had been before the transfer of the patient to Dr. Stevens’ care. The patient’s mother was much discouraged, and in justice to the fairness of the trial I was obliged, rather against my better judgment, to *urge* that further operations, etc., be allowed. The first decided improvement appeared thirty weeks after beginning of treatment; and any specialist who has had cases of chronic chorea (even of from three to six years’ standing) under treatment will recognize this as an absurd limit of trial of any one treatment. From that time, December, 1887, very little improvement occurred up to April, 1889. At times the chorea was worse, at times almost free from chorea, but never, even two years after the “beginning of treatment,” was the patient free from chorea. Since April she has been nearly free from chorea, except at time of menses. She is not *cured*.

In this connection it is well to bear in mind that the medicinal treatment, which in a first stage extended only over a period of three months (February to May, 1886), apparently gave little result, yet was followed in the summer of 1886 by spontaneous improvement. The patient was (accidentally) brought to my clinic at the College of Physicians and Surgeons in November, 1886, and was there treated by Dr. Berg with full doses of arsenic. This second course of treatment was followed by “improvement.” Consequently it may be stated that during the summer and autumn of 1886, and during the winter of 1886–7, improvement was steadily taking place; and that when the child was sent to Dr. Stevens, in April, 1887, she was in the full tide of spontaneous improvement, which is well known to take place in almost all cases of simple chorea. Yet, at periods of three and of fifteen months after putting on glasses, the chorea was *worse*.

I should add that during the whole course of the first treatment (February to May, 1886) I always had very grave doubts as to the exactness and faithfulness with which my orders as to doses of Fowler’s solution and as to rest were carried out. The rest was, I am quite sure, not enforced.

On the whole, I would express it as my deliberate opinion that the ophthalmic treatment of this case has had very little, if anything, to do with the great improvement, which did not begin until thirty weeks after the first

glasses were ordered by Dr. Stevens, and which has not terminated in a cure at the expiration of nearly two and a half years. During these twenty-nine months the patient has submitted to about 13 operations, and has worn seven or eight modifications of glasses before her eyes: a most extraordinary exhibition of patience and faith on the parents' part, and of perseverance and undaunted courage on Dr. Stevens' part.

No. 2.—Miss Flora K., age 19 $\frac{1}{2}$, nat., U. S., date April 22, 1887. M. Allen Starr.

No epilepsy in the family.

No insanity in the family. Father was in first stages of locomotor ataxia when she was born, is now in third stage. Mother living and healthy. This child is only one who has been ill. Two children died. One born dead and one died at nine months of pneumonia. Patient was born naturally (fifth child). Was well until ten months old, when she had measles. At age of one year had first attack, lay unconscious with eyes open, motionless for twenty-four hours. No convulsions. Then had fever, and for two months had "brain fever." For three months after this was unable to speak or walk. After this the spasms began just as they are at present; they have continued ever since at intervals.

She has two forms of attacks, (1) slight, a sort of faint, during which she is unconscious, but moves her hands; she does not fall or cry out, or bite her tongue; these last a minute, when she gives a cough, and it is over. Sleeps sometimes after these attacks for awhile.

(2) Typical attacks of grand mal; cries out, falls, froths, bites tongue, has general convulsions, during which she passes water, and after it sleeps for several hours. Wakes with a headache which lasts for some hours. Rarely has an aura from the stomach usually none. The frequency of the attacks varies greatly. During the early years of her life they were at times frequent, one to five a day. At times only at intervals of three to six months. At puberty they increased in frequency, and for the past four years, during which she has been under my care, she has varied from one to ten attacks monthly; the average for thirty months being five a month. During the past three months has had fifteen attacks, of which two have been severe. She has been treated chiefly by bromides for four years, zinc, borax, belladonna, antifebrin and nitroglycerine having proved useless.

Present Condition.—No tenderness of scalp or back. Has occipital and frontal headaches occasionally, not menstrual. Reads Snellen types xxx with either eye at twenty feet. Eyes move perfectly in all directions. No deviation apparent. No paralysis of face or tongue. Dynamometer 85 R., 82 L. hand. No paresthesia or anesthesia. Tendon reflexes normal. No ataxia. Taste and hearing good. Has cough, loud barking, with occasional expectoration; it sounds like the cough of chr. bronchitis, but lungs are normal. No cardiac murmur. Liver and spleen normal size. No abdominal tenderness. No palpitation. Slight dyspepsia at times, not constant. Bowels regular. No need of laxatives. Urine normal; no alb.; no casts; no swelling of feet. Menses began at 13 $\frac{1}{2}$ years; has always been regular except for one year, about eighteen months ago, when they would go over from three to six days; never

ceased; she is unwell four days; flow is slight. She has pain before the flow; not severe. No vaginal examination made. No leucorrhœa.

For past four years since taking bromides her memory has been weak, and now it is poor. She has no hallucinations; is not stupid. She has a chronic eruption on her face, papular, not purulent. Has headaches in forehead occasionally. Occasionally dyspeptic symptoms, with acid rising and wind from the stomach and nausea, which has subsided when medicine was stopped. No constipation; has fits of coughing at night with discharge of mucus. Slight spinal tenderness in upper dorsal region. Slight ovarian tenderness on right side.

M. ALLEN STARR,

Confirmed by Dr. DANA.

April 22, 1887.—Abduc. 4. Adduc. 8. Orthophoria.

R. V. $\frac{20}{40}$ — : $\frac{20}{20}$ W. + $\frac{1}{16}$. Ophthalmoscopically normal.

L. V. $\frac{20}{40}$ — : $\frac{20}{20}$ W. + $\frac{1}{60}$. WEBSTER.

June 9.—Notice of operation for hyperphoria.

June 10.—Nineteen fits during past six weeks since first examination,

April 22d. Operation performed to-day on sup. rect. of L. eye.

June 17.—No attacks for past week; has had headache for one week. Eruption and cough less.

June 25.—Attacks on 19th, 21st, and six on 2d.

Sept. 20.—Severe attacks have occurred of late only at night. Slight attacks, in which she does not fall, occur at all times of day, and are not followed by sleep. The girl and her mother say that the number of attacks is much less, but reference to the register refutes this statement. She has taken no medicine since May 10th. She says that formerly on stopping the bromide the fits were much more frequent; while at present the cessation of taking bromide has not resulted in an increase of the number of attacks. Has had headache all summer, and especially since her operation. Says they are more frequent and severe than ever before. Complexion is somewhat better, but she has a marked red papular eruption on forehead, both cheeks and nose. Her cough continues and is troublesome at night, when she raises mucus. She has worn glasses all summer, which have been changed from time to time. They produced headache at first.

Sept. 29.—Operation for esophoria. Abd. 5. Esoph. 1. Division of 1 inter., after which abd. 10°. Exoph. $\frac{2}{3}$ °.

Oct. 7.—Six attacks last week; two very severe. Headache severe for past week. Vomiting occurred for three days after last operation. No medicine was taken.

Oct. 22.—Severe attack on 9th; eight slight on two succeeding days. The record shows that her attacks occur in groups at intervals of two weeks, one set coming on three to five days after beginning of menses. In each set of attacks there are one or two severe, and several—three to nine—slight attacks. She has had no headaches for past two weeks. Cough is about the same. Eruption continues as before. Her face is brighter, and she looks more cheerful. She wears glasses all the time. For the past week the left glass has been made opaque by paper put over it, so that she uses only the right eye. She was operated on by Dr. Stevens about one month ago (Sept. 29th). The operation was not followed by any discoloration of the eye by blood.

Nov. 5.—Since last report eight attacks, one very severe with fall, and unconsciousness for two hours after it. The fall was so heavy that her sister who lives in the flat below heard it, and came up. Has had headache, which came on after glasses were changed. Was to have been operated on yesterday, but examination showed hyperphoria of only $\frac{1}{4}^{\circ}$, no esoph. or exoph., and no operation was done. Menses occurred on 29th, and lasted four days. The attacks preceded the menses. Has pain on left side of pelvis during period. Digestion is perfect. Papular eruption the same. Has coughed more lately than for some time.

Nov. 19.—Since last report thirteen attacks, one of which at night (on 13th) was very severe; her tongue was bitten and her head bruised, so that it pained her and is still tender. Her father counted six attacks on Sunday in addition to the severe one at night. Has had considerable headache, and after the fit on the 13th, vomited. Digestion good.

Nov. 21.—Orthoph. for distance, but eso. in accom. 7° . Abd. 4° . Has been wearing prism 3° , base out. Right int. cut, leaving abd. 9° . Exoph. 4° .

Dec. 12.—Attacks fewer. Has more cough. One severe attack. One attack was at night; did not eat before retiring. Had no headache of late.

Dec. 23.—On the 22d a stitch was put in l. int. rect. Cough is much worse. In a slight attack recently she was unconscious as long as it took the Elev. R. R. train to go from 65th to 42d Street

Jan. 14, 1888.—Little headache. Cough as before. Eruption slightly better under tar soap. Attacks on 2d.

Jan. 30.—No attacks since 2d. Has had nausea and vomiting several times lately without known cause. Cough somewhat better.

Feb. 10.—Operation for hyperphoria; stitch put in eye int.

Feb. 17.—Severe headache for some days. In recent fit fell and hurt her shoulder badly.

March 10.—Severe attacks, one very bad. Headache, occipital. Has worn glasses for past two weeks.

April 3.—For past three weeks has worn glasses with one (R.) eye completely covered, so that no light reaches the eye. For the past two months occasional hysterical tendency to cry and laugh, not noticed by her mother before. Has had much headache on top and back of head in past two weeks. Cough a little better. Nearly broke nose in attack.

April 25.—Dark glass over right eye removed on 19th. No glass till 23d, then new pair. Complaints of occipital headache, drowsy, cough slight. Digestion good. Eruption about same as ever.

May 23.—Glasses changed on 16th and again to-day.

June 6.—Last six attacks occurred during the night.

Summary of Attacks in past Year.—June 1, 1887 to June 1, 1888: Monthly 25, 5, 13, 16, 17, 13, 16, 5, 22, 20, 18, 19. Total 179.

Compared with July 1, 1884-July 1, 1885: under bromide treatment. Monthly 7, 4, 11, 6, 6, 5, 4, 4, 10, 4, 9, 4. Total 74.

The eruption is not a bromide one, for it has not varied since the bromides were stopped. She coughs about as before, and has much more headache than she formerly had. The total effect of the year's treatment has therefore been in every respect most unfavorable. She looks somewhat brighter in the face.

June 6, 1888.—Operation for 2° l. hyperphoria. L. sup. rect. cut, leaving r. hyperphoria $\frac{1}{2}$. WEBSTER.

June 15.—Operation said by Dr. S. to be successful. She is now to wear "stronger" glasses. No headaches. Cough worse. Great shock on 10th (death in family). Two bad attacks on the 11th. At present vomits with the attack, and often has stomach aura.

July 3.—Much nausea for past three weeks; less headache; but headache has been so bad for past two months as to lead her to have her hair cut off.

September 10.—Thirty attacks since last note; two very severe. Suffers much from nausea and vomiting, and has epigastric aura. Has less headache than in the Spring. During the Summer Dr. Boyer has examined her eyes twice a week, but she has worn no glasses, and had no operation. Since Sept. 4th she has worn new glasses.

Sept. 25.—Headache almost constant since Sept. 10th.

Oct. 10.—Fifteen slight attacks in four days. New glasses given on the 6th, and had twelve attacks on the 7th.

Oct. 24.—To-day wears two pair glasses, one of them new. Much nausea and vomiting of late. Eruption looks bad. Very severe fit on 21st.

Nov. 20.—More headache and cough than for some time. Glasses on r. eye changed to-day.

Dec. 22.—Two severe attacks in two weeks, and fourteen slight attacks. Wears glasses.

Jan. 10, 1889.—One severe attack on 2d. Has had less headache, but much nausea.

Jan. 17.—Note from Dr. Webster states a limitation of visual fields was noticed. Central vision $\frac{3}{8}$. Paleness of the optic discs. Probably incipient atrophy.

Feb. 16.—Patient has had twenty-two attacks since last note, one severe. Her general health is about the same.

Feb. 27.—L. hyperphoria $\frac{1}{2}$ °; no esoph.; no exoph. Adduc. 14°; abd. 6° STARR.

March 16.—Is wearing glasses 1° base out over each eye. She had nineteen attacks during February and nine in March, one of which on the 12th was very severe; bruises evident.

April 1.—Glasses changed on March 30th. On 31st she had two very severe attacks, hurting herself badly by falling. She still has headache, and vomits at times and coughs some; but she is fat, and looks well.

May 13, 1889.—Flora K., hyperphoria R. 1°; exophoria $1\frac{1}{2}$ °; abduction 6°. She has been wearing prism 1° base down over R. She is directed to go without glasses for a few days, and come again. WEBSTER.

June 1.—Number of attacks monthly from June 1, 1888 to June 1, 1889: 12, 13, 11, 6, 19, 17, 16, 22, 19, 11, 16, 12. Total 174.

Number of attacks monthly from June 1, 1885. to June 1, 1886: 4, 4, 3, 6, 3, 2, 6, 5, 3, 1, 7, 2. Total 46.

June 15.—On June 6th all glasses were removed, as patient had had seven attacks between June 2d and June 5th, several of which were quite severe, her parents hearing her "working" in bed. She went from June 6th to 15th without glasses; and to-day a new pair were put on. She has suffered from headache, vertigo, nausea, and occasional vomiting for some days.

June 29.—She has worn the new glasses constantly since last visit. She has had eight attacks, one only being severe.

Sept. 18.—She has worn the same glasses all Summer, and no operations have been performed. The attacks have been about as frequent as ever, but are somewhat lighter, and she has not fallen for some time. She has had headaches about once a week, and occasional attacks of vomiting. She has bled at the mouth once or twice lately. The eruption is the same. Digestion good. She looks fat and fairly well. Forty-six attacks since last note, three of them severe.

Oct. 2.—Notice of operation on the 4th.

Oct. 16.—Operation was performed on int. rectus of left eye, and one week later on int. rectus of right eye. Ecchymoses remain. She is to-day wearing prisms 1° l. 2° r. bases out. She now sees double on turning her eyes to either side. During September she had but nine attacks, the least number since 1887.

Oct. 31.—Abd. 7°; abduc. 15°. Exophoria 3° at 20'; 6° at 20". Crossed diplopia with red glass in middle and upper field when looking to the right.

W. O. MOORE.

Summary.—From July 22d, 1883, until April 22d, 1887, she has been under bromide treatment, and the records show that her average was about five attacks monthly, there being very few severe attacks (only four severe in eleven months of 1884 when record was kept with care and the character of all attacks recorded).

From April 22d, 1887 to October 13, 1889, she has taken no medicine whatever, and the records show that her average was about thirteen and a half attacks monthly, the severe attacks having increased in frequency (there were ten very bad attacks in twelve months in 1888).

During this period there has been no change in a severe erythematous eruption which had been ascribed to bromide; her nervous cough has continued at intervals as before, though on the whole it has troubled her less; and she has become subject to severe, and at times continuous, headache and attacks of nausea and vomiting. In spite of these facts, she and her mother affirm that she is better, and show confidence in the treatment. Her personal appearance and care in dress are improved, so that she looks well, and she has gained some flesh; but in all essential respects she is certainly much worse than under bromide treatment. The bruised appearance and the serious injuries caused by the fits had no parallel during the first four years of treatment, while the very great increase in the number of attacks of petit mal cannot but have an unfavorable effect upon her mind.

The ocular treatment has, therefore, after a patient and continuous trial of thirty months, utterly failed to ameliorate her epileptic condition, which has steadily grown worse. It has produced a condition of crossed diplopia when looking to the right.

ANNUAL REGISTER OF ATTACKS.

Case 2.

SEVERE ATTACKS X

SLIGHT ATTACKS •

1884	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	X	•
Jan'y.											••																						2
Feb'y.				X	X																											3	
March.													X																			1	
April.											••																		••			1	8
May.																••											X					1	3
June.																																?	?
July.																						X					••			X	2	5	
August.																																	4
Sept'r.			X								••																					1	11
Oct'r.																					X											1	6
Nov'r.										••	••	••																					6
Dec'r.										•																			••				5

Bromide Treatment.

1888.....	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	X	•	
Jan'y.	••	•																															5	
Feb'y.	•X	••	••	••	••	•											••	••														1	21	
March.	X	•	•	••	•												•X	•	•							••	••	•				3	16	
April.																•	••	••															9	
May.					•		••	••	••	•											X						••	••	•			1	18	
June.												•X	•	••											•	••	••	•				2	10	
July.										••	••	•																	X	••		1	12	
August.	••	•											•					•	•	••	••	•											11	
Sept'r.					X							••	•																				1	5
Oct'r.				•	•	•	•	•	•												•X	•				•							1	18
Nov'r.	•	•	••	••	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•		17	
Dec'r.						X	•	•	••	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•		1	14
1889.....	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31			
Jan'y.	X	•	•	••	••	•												X	••	••	••	••										2	20	
Feb'y.				•	•	•	•	•	•	••	•														•	••	••	••						19
March.	••												X	••	••	•															•X	3	8	
April.	•	••	••	••														•X	X	•	••	•											2	14
May.										X	•	••	••													•							1	11
June.	•	•	•	••	•												X	X							••	••	•						2	13
July.										••	••	•										X	••	••	••	•							1	16
August.							X	•	•	••	••	•														•	••	•	•	•	•		1	19
Sept'r										•	•	•					••	••	•															9
Oct'r.			•	••	X													•	••														2	11

Ocular Treatment.

No. 3.—John McGrane, age 14, epilepsy, Dr. George T. Stevens. Has been subject to convulsions since he was two years of age. Attacks occur generally once or twice a day, but he has intervals of two or three days, or even a week, between attacks; such an interval may occur once in four or five weeks.

Attack usually commences with a sense of dizziness, which lasts long enough for him to seek a place of safety or warn his friends. He then has convulsions of one or of both sides, more commonly of one side only. He becomes unconscious, and remains in this condition from one to four minutes; but at times the attack is very light. Generally bites his tongue or cheeks. Recovers fully after half or three-fourths of an hour.

Mother thinks that in half-dozen instances she has modified or prevented attack by application of cold water to head.

In the first years of his complaint his attacks were less frequent, but much more severe. At times he has remained in a series of convulsions for two or three days.

Has now habitually a sense of dizziness, and he is much troubled with headaches. Of late the pain in the head is less intense than formerly and the dizziness is greater.

He is habitually constipated, and has been accustomed to use laxatives since he has been epileptic.

Has been under medical treatment, but the mother cannot tell what medicines have been used.

(Signed)

GEO. T. STEVENS.

FURTHER NOTES BY DR. STARR.

Two other children have had convulsions up to the age of three. All children (four) have been very nervous. Father has attacks of cardiac pain very severe (angina pectoris?). Mother had attacks of migraine until age of twenty-two, and as a young woman was subject to "fainting spells."

The boy seems to have some control over the severity of the attacks. If he gets up and runs about at time of attack, it is much lighter, and he says himself that he can "stave an attack off." There is a mental aura, consisting of a sense of fear, so that he wants to hide away, and he often talks during an attack to some one who seems to him to be behind him and whom he never sees. The character of the attacks have wholly changed since his sixth year. Formerly the attacks were distinctly those of grand mal, with involuntary passage of urine. But for the past eight years he has not done this in a fit. The application of cold water never stopped the early fits. Now it does.

(There is evidently a great difference in the nature of the present attacks and the attacks prior to his sixth year.)

While for the past two months he has been having on- or two attacks daily, he has had none for the past six days since a change of physicians was proposed. (Is there some mentalelement here affecting the condition?) There is very marked phymosis, and he has never succeeded in retracting the prepuce. Denies masturbation, but a slight examination of the organ produced erection and sighing, and he knew what was referred to when questioned indirectly regarding masturbation. (Such phymosis has been known to cause such attacks, and several physicians have recommended circumcision in this case.)

No paresis or anæsthesia anywhere. Reflexes normal.

FURTHER NOTES BY DR. DANA.

The boy had eczema capitis, from a short time after birth, for nine months or a year.

Every month he has had a remission of a week.

Seven years ago he had a change of physicians, but got no better after it, or under the new treatment.

Right side of face is more innervated than left, and right palpebral fissure smaller.

Skull symmetrical.

The attacks involve only one extremity, lasting one to four or five minutes.

At commencement of treatment there existed homonymous diplopia, with ability to blend images when prisms equal to 17 degrees with their bases out were placed before the eyes. The tendency inward, as shown when a prism with its base down was placed before one eye, was 29 degrees.

There was constant oscillation of the eyes in a somewhat irregular manner.

The patient has hyperopic astigmatism, corrected by: R. E., S. + 1.75, with C + 2.00, at 90 degrees; L. E., S. + 1.00, with C + 3.00, at 90 degrees.

On May 9th, glasses were prescribed according to the following formula:

$$\text{R. S.} + .75 = \text{C} + 2.00, 90.$$

$$\text{L. C} + 3.00, 90.$$

On May 13th, tenotomy of the internal rectus of the right eye was made, relieving diplopia and leaving no manifest esophoria. On the following day, however, homonymous diplopia existed. In the three days preceding the tenotomy, the patient had four severe fits. (No daily record of the attacks previous to the 10th was kept.) On the 20th, 22d, and 31st of May attacks occurred less severe than usual. On June 6th tenotomy of the left internus was made.

The patient had a slight attack on June 7th. Between this date and June 24th three attacks occurred. The patient at this time was able to maintain binocular vision while wearing his cylindrical glasses. Homonymous diplopia, however, appeared at once if a red glass were placed before either eye.

A third tenotomy, this time on the right internus, was performed, immediately after which there appeared an excess of correction of about eight degrees. An hour later no excess appeared. Within a few days after the operation, homonymous diplopia reappeared, and during my absence, until about the 10th of September, nothing was done excepting that from time to time observations were made and recorded. During this time, from June 24th until September 24th, eleven attacks are recorded, some of which were very slight, and some of ordinary severity. On September 24th he had one slight attack, and between October 4th and 6th four severe attacks occurred.

During the latter part of June it became evident that associated with the homonymous diplopia was a certain amount of right hyperphoria. No action in regard to this was taken except from time to time to apply prisms with a view of determining the amount, until October 10th, when an operation for tenotomy of the superior rectus of the right eye, for the correction of right hyperphoria, of from two to five degrees, was done. Up to October 24th no further attacks had occurred.

It will thus appear that, during 167 days, twenty-two attacks—light and severe—have occurred, while, under circumstances similar to those preceding his ocular treatment, there would have been expected at least 167 attacks, or on an average one attack per day.*

During the time that he has been under observation there has been marked improvement in his physical and mental conditions. Notwithstanding the efforts that have been made to correct the ocular muscular defects, the patient is still subject to very important muscular anomalies, and has double vision a part of the time.

It has been interesting to observe that at such times as the muscular anomalies have appeared to be most nearly corrected, that the frequency of attacks have been proportionately diminished, and with a return of the tendency to diplopia the attacks have been more frequent.

(Signed)

GEO. T. STEVENS.

33 W. 33d Street, New York City,
October 27, 1887.

No. 4. George G. K., age 13, chorea; Dr. Seguin. First seen and treated at the Manhattan Eye and Ear Hospital; Nervous Department, February 25, 1887.

Was a healthy child. No convulsions. Present illness began as simple general chorea five years ago. A few weeks later, had a period of excitement—running around shouting, and lasting some weeks. Details not well recalled by friends. Never quite free from chorea since, though it was often reduced to a minimum. Was able to attend school at times. Was treated most of the time by several physicians. Time of appearance of electric form of chorea uncertain; perhaps two or three years ago.

Involuntary ejaculation of words began last autumn, and has persisted with intervals of freedom. Ejaculates single words or short sentences, usually obscene, in a perfectly involuntary and frantic manner simultaneously with choreic movement (examples of ejaculation), "damn it"; "mother's dead"; "prick"; Theresa has a fit" (an older sister has chronic epilepsy), etc. This constitutes coprolalia (Charcot). Echolalia not observed. Choreia consists in very irregular muscular jerks varying in extent and distribution at different times, always, however, single jerks or discharges shown by extension movement of arms, nodding or rotation of head, twitch of naso-buccal muscles.

* An exception must be taken to this statement, for it is recorded in the history that he had gone six days prior to the first operation without attacks, and that he has intervals of two or three days or even a week between attacks.—M. A. STARR, October 28, 1889.

Almost never jerks legs. Trunk muscles somewhat involved as shown by bowing movement.

Ejaculation of formed sounds (words) are rare in last two months, but always gives a cry or grunt representing a clonic action of laryngeal muscles.

Causes. None known. An injury to back of head with partial loss of consciousness occurred three years ago. Masturbation excluded with reasonable certainty. Only case of neurosis or "nervous trouble" in family is elder sister, now aged thirty-six years who has been epileptic since scarlet fever at eight years.

Treatment. Has had a great deal of medicinal treatment, nature unknown.

Under my care since February 25, 1887. Has had successive trials of solution of chloro-phosphide of arsenic, and of hyocyania $\frac{1}{2}$ grain, three or four or five a day. This remedy produced a marked improvement for a few weeks. Rest always seems beneficial

Physical Examination. Electric chorea as above, irregular single jerks of muscle-groups in neck, trunk, arms, and face, and of laryngeal muscles. The diaphragm apparently involved sometimes. No paralysis or anæsthesia. Heart normal. Pupils equal and normally active. Marked anemia. Organic functions well performed.

Occasional headaches from the first, at times very severe. Occasional vertigo on sudden exertion after a period of quiet.

(Signed)

E. C. SEGUIN.

Confirmed by

M. A. STARR.

May 20, 1887. After examination by Dr. Starr the patient is sent to Dr. Stevens. Utters grunts and cries with choreic jerks, but coprolalia has entirely ceased.

No formula of ophthalmic examination furnished.

May 25. Note from Dr. Stevens. "We will operate on Geo. F. K. (Dr. Seguin's patient) for esophoria, on Friday, May 27th, at 1:30 P. M."

June 3. Note from Dr. Stevens. "We will operate on Geo. F. K. (choreic) for esophoria, Monday, June 6th, at 1:30 P. M."

June 8. Note from Dr. Stevens. "I see no objection to allowing a little quinine to K. He thinks he is rather better; perhaps he is." Pil. quin. sulph. gr. iij *ter die*, was ordered by me.

June 25. Note from Dr. Stevens. "Dr. Stevens requests me to say that he will operate on —, on Geo. F. K. (patient of Dr. Seguin) for esophoria on Friday, June 24th, at 1:30 P. M."

October 11. Note from Dr. Stevens. "There will be an operation on Geo. F. K. (choreic patient of Dr. Seguin) for hyperphoria, on Friday, October 14th, at 1:30 P. M."

October 4. Note by Dr. Webster. "I saw Dr. Stevens operate upon young K., Dr. Seguin's case of coprolalia (?) to-day. The boy had had previous tenotomies of his right superior rectus and both interni. The condition immediately before to-day's operation was: abduction 6°, esophoria 0°, right hyperphoria $\frac{1}{2}$ °. Dr. Stevens did a partial tenotomy of the left internus to-day, immediately after which there was: abduction 8°, exophoria 1°, and hyperphoria 1°."

October 22. Presents substantially the same chorea and laryngeal sounds. Last night a couple of bad words came out, "damn it," etc. He says: "I felt it coming but could not hold it back." Claims to have been better, or nearly free from symptoms for a week without "grunting"—several times in the summer. At times was two or three days without chorea (?). Choreic movements are less extensive. General condition excellent. Was better (of chorea) while in the country at play. Operations have been done on the 14th and 21st of this month (no notice sent).

December 5. Note from Dr. Webster. "I was at Dr. Stevens' on the 3rd and witnessed the following: Geo. K., exophoria 2° , abduction 10° . Advanced left internus, leaving esophoria 4° and abduction 2° ."

December 9. Mr. C. (father of Elizabeth C., case 1) voluntarily states that he has recently heard the coprolalia boy grunt in Dr. Stevens' office; not as loud as formerly, but very audibly, so much so that one morning, not long ago, he badly scared a lady in the waiting-room.

December 22. Presents same symptoms, viz.; sudden electric choreic jerks of neck and shoulders with occasional slight jerks of hands. With a great many jerks he grunts ("ong" or "eng" with French sound). Jerks occasionally extend to the legs (flexion movements of knees). I could hear him in my reception room, separated from me by closed solid doors. He states that since last call he has not had five hours without grunts. Has less of it while busy or walking about. No bad words since last visit.

December 30. Choreia worse, with frequent grunts. While sitting has same movements as formerly, viz.: a nodding or flexion jerk of arms; usually with a grunt, sometimes with an inspiratory laryngeal sound. The force of the movement extends to the legs. While walking has distinctly right hemiplegic jerks of arm and hand, and of leg: the foot is made to strike the floor with its anterior extremity. The patient says he "stubs his toes," and has worn the front end of the sole of right shoe more than the left. There is no drooping of the shoulder or dragging of the right foot as in ordinary hemiparesis. The choreic motion is both more violent and more frequent; tongue deviates to right. [This condition is 28 weeks after transfer of patient to Dr. Stevens.]

1888. February 17. Much chorea and grunting this morning. I heard him in my reception room through closed solid doors. Claims that he has some days without grunts. Says that bad weather, pork, and pickles make him worse. Nothing has been done to eyes since the end of December. Wears spectacles constantly. Says that right arm jerks most; tongue goes to right. General condition good.

February 24. Note by Dr. Moore. "Geo. K., patient of Dr. Seguin. Hyperphoria right 2° . Tenotomy of right superior rectus. After, hyperphoria 4° ."

March 16. "Another operation done on right superior rectus about three weeks ago; first this year. Since the 14th, left glass of spectacles covered with translucent plaster. Has about the same amount of chorea. This morning it is universal, but more marked in right limbs; most of all in head and neck. Does not grunt just now, but has done so this morning."

April 20. Note by Dr. Moore. "Exophoria 3° . Advanced by a single stitch the left internal rectus, producing esophoria 16° ."

May 4.—An operation done two weeks ago. Is very anæmic, and suffers from rheumatic (?) pains in right hip, and has some herpes labii. Chorea unchanged; rather worse to-day than on average; no grunts. Patient says he seldom grunts now, but just at this moment makes a slight grunt.

June 1.—“Two weeks ago (about) had an operation performed, and after it grew rapidly more choreic and ‘grunty.’ On Tuesday (29th May) Dr. Stevens ‘took a reef’ in the left external rectus, and some improvement soon followed. Is now more choreic, and grunts more than for many months. Denies extraneous causes of aggravation, such as fright, ill-health, etc. Movements are as before, perhaps more head movements. Speech distinct. Tongue deviates to right; grasps of hands equal.” [It is to be observed that the choreic movements are about as bad as ever, one year after beginning of treatment.]

Nov. 4. Note by Dr. Webster. “K., the coprolalia patient, showed hyperphoria 2° right eye; esophoria 1° to 2°, and abduction 5°. Dr. Stevens advanced the left superior rectus just enough to correct the hyperphoria (of the right eye, and about $\frac{1}{10}$ ° more. This operation was rendered necessary on account of an over-correction of left hyperphoria by a recent tenotomy.”

Nov. 28.—“About three weeks ago, after an operation, symptoms increased a great deal; loud explosions of laryngeal sounds, like a mixture of ‘ha’ and ‘hein,’ accompanied by jerks of head, shoulders, arms, and occasionally legs (only right leg). Looks pale and miserable, from a ‘cold’ which developed two weeks ago. Had fever and some sore throat, for which he was in bed several days. Has ‘explosions’ every few moments while talking with me. Much less when alone and when reading. I recognized George’s presence from the second floor of my house to-day as he came in by his noises. No words now escape him. Is free from ordinary chorea.”

Dec. 3.—Note by Dr. Moore. “Exophoria 2°. Advancement of left internal rectus, leaving abduction 2°, and esophoria 4°.”

Dec. 7.—An operation done on left internal rectus on Dec. 3d. Worse since. On 5th had severe right supra-orbital neuralgia. To-day worse than last week. Extensive jerks of face, head, and whole body. Sudden electric jerks, with a cry or semi-hiccup. While waiting in my hallway was very noisy. No words ejaculated. Admits great irritability. General condition better.

Feb. 6, 1889 (about).—Is much better; presenting but little chorea, and seldom crying out or grunting. A formerly used pair of spectacles, was put on a week ago.

March 1.—About Feb. 10th caught a severe cold, with sore throat, alveolar abscess, etc. Great increase in chorea followed. Is now pale; has large choreic clonic movements of face, neck, and arms, with quite loud grunts. Mouth opens wide when the cry comes. Is almost as choreic as when transferred to Dr. Stevens. This is the second time that the event of an inter-current illness has been followed by marked aggravation of the chorea.

George thinks that he has had in all nearly twelve operations done on his eye-muscles. He has had four different sort of spectacles, besides frequent changes in their arrangement and order of use.

March 19.—Hyperphoria R. 3°; abd. 7°. Has worn 3° prisms for weeks. Operation: tenotomy of R. sup. rectus, after which no hyperphoria.

W. MOORE.

March 27.—Operation on R. ext. rectus on 21st. Has jerked more since, and to-day shows large hard jerks of neck, arm and trunk, with grunts, which could be heard at a distance. "Cold" gone, but is still pale. Has a bad herpetic sore (double) on l. lower lip. Ord. ung. acid carb.

April 24, 1889.—Has been much better for one month. He has had some days without a single sound. Has had diplopia when looking off to his right. New glass put on yesterday corrects this, but patient speaks of them as very strong, and thinks he is worse this morning. Makes a few whoops or grunts, and shows a good deal of choreic large movements of the right arm, both legs and neck (very little of last). Speech good. General condition good. Has grown a good deal.

May 11.—"I saw Geo. K. to-day. He was barking at frequent intervals, but not so loudly as he used to, and the excursions of head and upper part of the body were much less extensive than formerly. He is wearing prism 2°, base out.
D. WEBSTER."

Oct. 22, 1889.—Had much chorea until end of August, after that was much better. About the middle of September an operation was done on his left internal rectus; for two weeks he was worse; then began to improve. Last week he was very well; several days without grunts. Another operation was done on his left external rectus on Oct. 19th. The eye is still sore. To-day, although much better than when last seen (in June), he grunts pretty frequently and has choreic jerks of shoulders and hands; legs quiet.

Oct. 31, 1889.—"Dr. Stevens sent me Geo. K. this morning, and I made the following notes. Dr. Stevens has operated upon his eyes several times, and George thinks he is much improved in several ways. He sleeps better, feels more lively, has fewer headaches, doesn't throw out his right foot and right arm as he used to, and does not bend over nearly to the floor as he used to. He still whoops very often, but not nearly so often as he used to. *He sees double when looking far to the right*, but says that does not annoy him any. In the general use of his eyes for reading, etc., he is no worse than before operations were commenced. He has a sandy feeling in his eyes at times, due to slight conjunctivitis.

V. $\frac{2}{15}$ — E. both. Hyperphoria L. $\frac{1}{4}^{\circ}$; exophoria $\frac{1}{2}^{\circ}$; in accom. 1° ; abduc. 7° ; adduc. 30° ; sursumduc. R. $\frac{3}{4}$; L. I. Fields, roughly tested, seem normal. Ophthal. appearance normal.
D. WEBSTER.

Summary.—This case of chorea has not been cured or much relieved by nearly two years of the ocular treatment. The patient has shown truly extraordinary patience and perseverance in the pursuit of a cure.

When sent to Dr. Stevens, medicinal treatment (by hyoseyama) had entirely removed the coprolalia, and somewhat diminished the chorea. The coprolalia reappeared while the lad was under Dr. Stevens' treatment, viz., Oct. 21, 1887, four months after beginning of treatment. At periods of twenty-eight weeks, and of one year from beginning of treatment, it is noted that the chorea is as bad as at first. This case is at present improved, but is in no sense cured. He has diplopia as a result of treatment.

No. 5—Aggie H., age 13, epilepsy; Dr. Dana, through Dr. E. D. Fisher. Parents healthy; one aunt hysterical; family history otherwise negative; natural birth at full term. Between the 6th and 24th month she had from 6 to 10 convulsions, then they ceased entirely. The present disease began at the age of 11 years without known cause.

She suffers from attacks of *petit mal*; she has no spasmodic movements and no attacks of *haut mal* at all; she has had as many as 6 to 10 daily; she is now having 2 to 4 daily; she is fairly well grown and intelligent, with no physical deformities; skull is symmetrical and of average size. Diagnosis confirmed by Dr. Starr.

History of ocular conditions.—Examination by Dr. Moore, Jan. 3, 1888, showed: Abduc., 4° ; Add., 12° ; orthophoria.

The same condition was found by Dr. Webster, Jan. 4th.

Dr. Stevens thought that she had latent hyperphoria, and on Jan. 4, 1888, he operated, performing a partial tenotomy of her right internus, dividing the tendon at its attachment, causing at the time, exophoria, 7. Next day there was abduction, 8° ; esophoria, 1° .

There is notice of operations to be performed on Nov. 19, 1888, and on Feb. 10, 1888, but no ophthalmologist's records.

On Nov. 26, 1888, Dr. Webster found: esophoria, 2° to 6° ; abduction, 5° .

On Jan. 22, 1889, Dr. Webster found: esophoria, 5° ; abduction, 6° ; on this day Dr. Stevens did a resection (modified advancement) of the right externus, leaving her with: exophoria, $1\frac{1}{2}^{\circ}$; abduction, 10° . March 16th, notice of operation on March 19th, for esophoria.

March 19th, 1889: esophoria, 5° ; abduction, 4° ; operation on L. E. Resection of tendon of left externus (so-called by Stevens), really an advancement of the externus. After operation: exophoria, 1° , with abduction 9° .

This patient is said to be better by Dr. Stevens, who claims that after the first operations, the "attacks, 50 or 60 in a day," were reduced to 6 or 7 daily. The mother thinks she is better. This patient has strabismus convergens, as her eye turns in most of the time.

History of the seizures.—On Nov. 26, 1888, the mother states that the patient had had only 3 or 4 seizures daily since the operation in Jan., and that the seizures are milder; she thinks the child better.

On Jan. 22, 1889, the case is said by the mother to be improving. The child still has the seizures every day.

May 13: esophoria, 5° , and abduction 5° . She is wearing prism 1° base out over each eye. Tenotomy of right internus was done to-day, leaving orthophoria and abduction 8° .
D. WEBSTER.

Sept. 17, notice of operation.

Oct. 29. The girl is having from 1 to 3 attacks of *petit mal* daily, and cannot, therefore, be said to be in any marked degree improved, as at the beginning of treatment she was having from 2 to 4 daily.

No. 6.—C. D., female, age 9, epilepsy, Dr. Stevens.

General History.—Was a healthy child until the age of three years, when she had whooping cough very severely, which lasted several months. First attack of epilepsy occurred after a cough, had continued about a month and

was very severe, lasting about four or five minutes. About a month later had a second attack, then soon another. From that time there has never been an interval of a month between attacks; these having occurred during the past two years about once every other day, and for the past three months the average is more than once a day. Petit mal occur also from once in a few days to many times in the same day. Before a regular attack sometimes experiences pain in the stomach, but this is so habitual a complaint that it may be said there is no warning. In attacks there are convulsive movements at first. Often the attack is ushered in with a scream; sometimes says she is frightened. Attacks vary in duration from two to ten minutes. After attack sometimes recovers quickly and at other times is drowsy.

At seasons varying, but averaging about once a month, is in a state of profound dementia, which continues from ten days to a fortnight. During the more lucid interval is not bright, but has a sort of infantile simplicity; and answers questions within her comprehension correctly. Is feeble-minded at best. Walks with a sort of stagger, and "interferes," the toe of one foot coming in contact with the heel of the other. Very awkward in all her movements. She calls letters correctly, but cannot read common words of a few letters.

Is habitually constipated and takes drugs for the trouble. Has much dyspepsia. Has been under treatment by physicians in this country and in Europe.

Diagnosis confirmed.

C. L. DANA, M.D.

April 20, 1888.

C. D., Hy. R. 3. Tenot. R. sup. rect. after has $\frac{1}{2}$ over correction.

W. O. MOORE.

Nov 7, 1888—"C. D. was taken to the country in July. Her aunt, in whose charge she has been, called on me in September; said that the child was greatly improved mentally and physically. She asked my advice about bringing her to the city in September, and it was decided that she should come in October. She did not, and I have not heard further from her.

GEO. L. STEVENS."

No. 7.—E. F., male, age 14, chorea or tic convulsif; Dr. Stevens. Had at the age of two years frequent convulsions, which occurred in considerable numbers until he was five years of age, when convulsions ceased and he became choreic. He has continued to have chorea up to the present time. The choreic movements are of the so-called electrical type. They are mostly confined to the head. The face jerks, eyes snap, and he utters at times short and sharp vocal sounds resembling the bark of a dog. At times in the course of his complaint these barking sounds have been a conspicuous feature of his condition. At present these sounds occur somewhat rarely.

Is and has been accustomed to have severe headaches. Has had sensations of numbness of hands and feet.

He has been under the treatment of several physicians, with no material relief.

I have seen this patient, and confirm the diagnosis of chronic chorea so-called or tic convulsif.

C. L. DANA, M.D.

W. R. BIRDSALL, M.D.

April 20, 1888. Hyperph. R. 3. Tenot. R. sup. rect., and afterward 1 hyp., L. E.

(Signed) W. O. MOORE.

Nov. 7, 1888. "The chronic boy has been in town for a few hours on two or three occasions. I have been unable to arrange for him to come at a definite time, and will do so soon if possible. I think that I can arrange it.

GEO. L. STEVENS."

Nov. 26, 1888. Esophoria, 4°; abduction, 9°. Although he manifested no hyperphoria, yet that condition was supposed to be latent and to account for the apparent discrepancy.

(Signed) D. WEBSTER.

No further notes have been sent to the Secretary.

No. 8.—G. H., male, age 10, epilepsy; Dr. Stevens. Was first child. Has large head, well formed. As an infant extremely nervous and sensitive. Intellect unclouded. First convulsion at the age of three, followed during the next three years by about three convulsions each year. In each of these, eyes were set or rolled to left, pupils dilated, head turned to left, limbs in convulsions, grinding of teeth and foaming at mouth. After one or two of these attacks patient appeared for a time to be lifeless; pulse and respiration ceased. During past six years has had somewhat frequent attacks of much less violence. Becomes in these attacks unconscious, and when partial consciousness returns talks wildly, swallows violently, has violent twitchings of the extremities, and pupils dilate. These attacks are usually controlled by an anæsthetic (chloroform). Other attacks occur resembling epileptic vertigo, lasting but a few minutes, and usually occurring three or four times in the twenty-four hours. Such a succession of attacks occurs once in three or four weeks. Then an interval of some weeks follows.

Father thinks that if these premonitory attacks are not controlled by the anæsthetic, they will usually result in the more severe form of convulsion. These lesser attacks leave the patient weak and pale for two or three days, and inclined to sleep much. Goes into profound slumber after each convulsion. From the age of six until nine was free from the more severe form of convulsion, but after being sent to school for a few days a convulsion of great violence and duration occurred. The last great convulsion was in June, 1887. Is active and wide-awake, but pale, and lacks endurance. Memory is excellent. Has taken bromides and strychnine regularly for five years past.

Diagnosis confirmed.

C. L. DANA.

My understanding of the history was, this patient has very severe attacks of *haut mal* rarely.

Peculiar "premonitory" attacks every one to three weeks.

Ep. vertigo or *petit mal* several times daily.

C. L. DANA, M.D.

Patient seen at Dr. Stevens' office April 4, 1888. As the grandmother related the case to me, patient had his first convulsion, as stated above, at the age of three; the next attack was about one year later; the next, six months

later; six months later, another. For the next few years attacks were less frequent, not more than three or four, and last attack, as stated in notes, was in June. These are the convulsive attacks described. The so-called premonitory attacks, according to the boy's own description, are: First a feeling of nausea; at this warning he goes and lies down; slight vertigo accompanies it; then he feels sleepy, and goes to sleep, as he says. It is on waking that his ideas are confused and in which he talks incoherently. Grandmother states positively that convulsive twitching never occurs in these attacks, though he is confused and restless after his sleep. She states that after he lies down the face becomes quite red, and thinks that a pale stage (at the time he feels the nausea) precedes it. She thinks the grand attacks would occur if medicine were not given; but only one attack, the last one, has followed the attack of vertigo. This did not take place until the day following the premonitory attack. Anæsthetics have not always been used, but other medicines instead, she knew not what.

Diagnosis of epilepsy confirmed, the so-called premonitory attacks being in the nature of epileptic vertigo. The period of incoherent talking being a post-epileptic phenomena.

W. R. BIRDSALL.

April 12, 1888. Esoph., 8°; abd., 5°; tenot., 1, externus; after which abd., 12°. Esoph. still 4°.

December 3, 1888. Abd., 4°; esoph., 5°. Tenot. r. int., leaving esoph., 2° to 5°; abd., 9°.

Notes made May 25, 1888, by Dr. Birdsall:

Grandmother states that he has not had any convulsions, nor any of the attacks of vertigo (premonitory spells) since March 28th. Patient himself states that about April 27th he had one day the feeling that he was about to have an attack; resolved that he would think of something. It did not come on, and none has occurred since. Tenotomy was first performed April 6, 1888.

November 7, 1888. "The bright lad, from the western part of the State, with epilepsy, was in town for a day about a month ago. I did not know of his coming until he was here. I expect him again soon, and will try to have you see him. He was seen by Dr. Birdsall just before the summer vacation.

"GEO. L. STEVENS."

March 3, 1889. "Patient's general appearance is better than when last seen; looks as if he had increased in weight. He states from memory that he thinks he has not had any premonitory attacks, but has had one severe fit this month.

"W. R. BIRDSALL."

No. 9. David Sh., aged 12 years; epilepsy; Dr. Starr. No family history of epilepsy or insanity. Natural birth. First fit at age of fifteen months, lasting three hours when teething. After that he occasionally had "fainting spells" lasting five to ten minutes. When five years old these increased in severity and frequency. At the age of nine, regular severe general convulsions commenced, with biting of tongue, fall and cry, and followed by sleep, and these have continued ever since at varying intervals, sometimes as often as two in a week. In addition to the attacks of grand mal he also has the

"fainting spells" not attended by convulsions (petit mal). He has no aura of any kind prior to the attacks. He is a bright boy, has never had headache, has had no bad habits, has a good memory and is in good health. He is constipated and his mother is sure that when his bowels are not moved daily he is more liable to attacks. Physical examination negative.

Records of attacks from October 24, 1887, to April 28, 1888. November, 1887—3 severe, 3 slight attacks; 5 visits. December, 1887—5 severe, 2 slight attacks; 5 visits. January, 1888—record lost; 1 visit. February, 1888—1 severe, 5 slight attacks; 2 visits. March, 1888—2 severe, 5 slight attacks; 3 visits. April, 1888—1 severe, 3 slight attacks; 3 visits.

During this period patient was taking bromides of potash, soda, and ammonia, in doses varying from thirty to sixty grains daily. All medicine was stopped on April 23d.

STARR.
DANA.

Examination of eyes, April 23d:

R. V. = $\frac{3}{8}$ Hm. 0.75 D. Ophthalmoscopically
L. V. = $\frac{3}{8}$ Hm. 0.75 D. no lesion of either fundus.

No hyperphoria; esophoria $\frac{1}{2}^{\circ}$; exophoria in accom., 9° ; abduction, 6° ; adduction, 20° . R. 1. L. 1.

DR. WEBSTER.

May 2. Notice of operation.

May 4. Examination by Dr. Stevens. Right hyperphoria, $3\frac{1}{2}^{\circ}$; esophoria, 7° ; tenotomy superior rectus; after which, left hyperphoria, $\frac{1}{4}^{\circ}$.

October 31. Reports by request. Mother states that he attended regularly until the middle of September when, as the fits had constantly increased in frequency and severity, she had ceased to attend. During the summer he has been operated upon several times and has worn glasses constantly, but has taken no medicine.

Records of attacks from April 28th to September 4th: May, 1888—11 severe, 6 slight attacks; June, 1888—2 severe, 7 slight attacks; July, 1888—6 severe, 2 slight attacks; September, 1888—11 severe, 4 slight attacks.

CASE 9.—ANNUAL REGISTER OF ATTACKS.

Severe attacks, x.

Slight attacks, .

1888....	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	X	.
Jan'y.																																	
Feb'y.						X																				1	5
March.						X	.						.								.				X	.						2	5
April.						X	XX	XX	XX	.												.		.		X						1	3
May.						X	XX	XX	XX	.					.				X	X		.	.			X			.			11	6
June.		.	.	.					X									X	.	.	.											2	7
July.			X	XX	.													X		XX												6	2
August.	X					X		X											XX	.	.										X	6	3
Sept'r.	.	X	X	.		X		X	X	X	X	X	X						XX		.		.									11	4
Oct'r.			XX	X																													
Nov'r.																																	
Dec'r.																																	

The mother was urged to return to Dr. Stevens, but refused absolutely. The boy's general health is about the same, but during the summer he has complained frequently of headaches—not present before.

The result of stopping the bromide on April 23d was the occurrence of nine

severe fits between May 6th and 9th. Seven of the severe fits in Sept. occurred within five days, at the time of his father's death. But even eliminating these two factors from the record it is evident that under the ocular treatment the fits were twice as frequent, and during this treatment there was no tendency to improvement.

1889. October 30. He is now under Dr. Jacoby's care. During September, 1889, he had two bad and two slight attacks; October, 1889, he had two bad and three slight attacks. He now has

R	25
V	—
L	15

Adduction, 19° ; abduction, 7° ; esophoria, 4° ; left hyperphoria, $\frac{1}{2}^{\circ}$.

No. 10.—Eva S., age 16, native U. S., physician Dr. Fisher; complaint, chr. chorea.

Family history good. Well until three years ago. Sudden onset of chorea, after trouble in family. No rheumatism. No cardiac. Only cause known is emotional shock. Condition has varied; at times severe, at times slight; but continuous for three years in spite of treatment by arsenic, etc. At present moderate chorea r. > 1 . face not involved; neck jerks; tongue has jerked, but does not now jerk. No cardiac. Headaches. No dyspepsia. Constipation. Menses every three weeks seven days. Slight leucorrhœa. Menses began one year after chorea. No tenderness of spine or abdomen. Anæmia.

Ocular Exam. (Dr. Webster) May 4th.—Hyp. R. $1\frac{1}{2}^{\circ}$. Exoph. in accom. 4° . Abd. 5° . No esoph. No exoph. Myopic. astig. R. $\frac{2}{10}^{\circ}$ +. L. $\frac{2}{10}^{\circ}$ +.

May 25.—Notice of operation on 28th.

June 15.—Improved.

“ 25.—Still better.

July 25.—Relapse, but not so severe.

Aug. 16.—Almost quiet until menses, when again worse.

Sept. 11.—Movements returned; lasted only one week.

Oct. 10.—Operation on left eye.

Oct. 11.—Improved.

Nov. 11.—Tremor seems to involve muscles in usual situation, viz., upper extremities and shoulder and neck. Lower extremities also affected. Two patches of proriasis noticed on legs above knees.

FISHER.

Nov. 20.—Has less headache, but still has sharp pain in left temple at times. Menses irregular usually seven to eight days before time. The chorea is better, but still persists; greater on right side than on left. Upper lip twitches, and there is an expiratory sound in throat at times, which is said to have been worse. Both arms twitch and also the muscles of the neck. Legs twitch slightly. Knee jerks equal. Is wearing double glasses.

STARR.

Dec. 5.—Exophoria 3° . Abduction 10° . Operation. Left internus advanced, leaving esophoria 4° .

D. WEBSTER, W. O. MOORE.

Jan. 22, 1889.—Exophoria 4. Abduction 9. Operation. Resection (modified advancement) of the right internus, leaving orthophoria and abduction 6° .

D. WEBSTER.

Feb. 17.—Slight chorea in extremities, better than at last visit; r. greater than l. Slight noises in her throat frequently. There is no esophoria or exophoria nor hyperphoria, but there is a slight apparent int. strabismus, and abduction is 10° , while adduction is 19° . Is to have another operation this week. The chorea is slighter than it was at the beginning of treatment, but has not disappeared, and has advanced to the larynx, which was not formerly involved.

March 19.—Orthophoria. Abd. 6° .

W. O. MOORE.

April 24.—The jerking is slighter, but visible in neck, hands, arms, legs and feet r. $> l$. She was much worse for two weeks after last visit; jerked constantly. Then Dr. Stevens operated on the left eye, and subsequently the jerking became less. She has been taking medicine for psoriasis, which still affects her, there being large spots of psoriasis over her limbs and body. She has suffered from abdominal pains, with constipation, and is at present quite anæmic. Is wearing two pair of glasses at present. No eso. or exophoria, but slight $\frac{1}{2}^\circ$ r. hyperphoria. Referred to Dr. Elliot for treatment of psoriasis, with request that no arsenic be used.

Oct. 2.—She has been wearing the same glasses all summer. No operation. The chorea has varied; has never been absent. For the past four weeks has been more nervous, and at present twitching is present in all extremities, right side greater than left; and occasional sound in larynx is made. Psoriasis is very bad. Has had headache during the summer. Is now pale and thin. She does not think she is looking well, and is not. No medicine has been given for psoriasis.

Nov. 2, 1889.—“Report on the case of Miss S., whose eyes I examined to-day: She is wearing

Rt. + 2.75 D. c. axis 105° .

Lf. + 2.75 D. c. axis 75° .

and with these glasses her vision is $\frac{20}{30} +$.

R. V. = $\frac{20}{30}$ —: $\frac{20}{30}$ — with + 2.25 D. c. ax. 105° .

L. V. = $\frac{20}{30}$ —: $\frac{20}{30}$ — with + 2.50 D. c. ax. 75° .

Orthophoria with or without her glasses. Abduc. 6° . Adduc. 36° .

Ophthal. No lesion observed.

The patient states that she has had six operations upon her eyes, and that she is unimproved. She has no diplopia or asthenopic, or other trouble with her eyes. In short, she does not complain of her eyes at all.

D. WEBSTER.

Summary.—No improvement whatever after eighteen months of continuous treatment.

M. A. STARR.

No. 11, Miss L. P., Chorea; Dr. Stevens.—Has violent twitching of arms and legs. Eyes blink continually. Never looks at an object for more than a moment at a time. Arms wave about in wild gesticulations. Each hand moves up alternately as if to brush her short hair from her forehead; is continually disarranging her hair and garments with her movements. In walking she moves rapidly forward a short distance and then suddenly stops as if hesitating, (arms and eyes in continual motion) and then again advances. In talking her speech is characterized by the same irregularity and extravagance that is part of her general movements. When spoken to, although she grasps your question in-

stantly she hesitates, saying: "Sir," "Sir," and then suddenly breaks out with a verbose answer, correct and rational, but rapidly and hysterically, giggling at every opportunity and withall manifesting decided intelligence and quick perception. The trouble began three years ago while at school. She suddenly began to be irritable both mentally and physically. This was noticed by herself and family, but she was allowed to remain at school until eight months ago, when her restlessness and irritability became so marked that her parents were obliged to remove her from school. Since that time has gradually become worse until the present time when she appears as stated above. The presence of her mother is a continual irritation to her. She speaks sharply and irritably whenever her mother addresses or speaks of her. Has lately grown to dislike everything—music, studies, people, etc., that she formerly took most pride in and cared especially for. Heart's action somewhat irregular, but otherwise normal. No organic lesions discernible by ophthalmoscopic or other examinations.

The above corresponds with the account given to me and with the symptoms as observed by myself.

C. L. DANA, M. D.

July, 1888.

Nov. 7, 1888.

"The chronic girl has been in the office from time to time, but so irregularly that I did not think worth while to notify you when she might be expected. She has of late made three visits at appointed times and I think that she may now be relied upon to keep an appointment. I will endeavor to have her here (I think she will come, without doubt) on Monday, at one o'clock, or on another day if it will be more convenient to you."

GEO. T. STEVENS."

May 3 1, 1889.

Miss L. P., æt. 18, was seen by me at Dr. Stevens' office, at her *final visit*. She had been under Stevens' care a year; was operated upon for hyperphoria several times, and now shows Hyperphoria 0°, Exophoria $\frac{1}{2}$ ° and Abduction 9°, Dr. Stevens stated in her presence that when she first came under his care she was affected with chorea of the most violent type. She was unable to hold her water, and had great difficulty in getting a boarding-house on that account. She would urinate while at meals, in bed, and in Dr. Stevens' waiting-room and office. This part of the trouble is *entirely well*. She still has some chronic movements, but expresses herself as a *hundredfold better*. "An entirely new girl, in fact," than she was. She then *hated* everything: She now enjoys life and returns to her home very happy.

Dr. WEBSTER.

No. 12.—Gertrude E. W., aged 24 years, epilepsy. Dr. Dana.—Father died of phthisis, mother has migraine, sister has migraine; no epilepsy in family. Well until age of 19, when fright caused present attacks. Menses at 16, regular. At first developed slight seizures, which increased in frequency until in fall of 1888 she was having two or three a day. During the attacks she loses consciousness, has spasmodic twitchings of face and arms, would fall if not seated, has headache and sleeps afterward. In summer of 1888 had four severe convulsions, during which she bit her tongue. Since epilepsy began migraine has stopped. Good physique, fair health, some constipation, digestion good, courses regular

pupils large and mobile. She is now having petit mal two or three times a fortnight, usually about time of menses; had one attack last week; was unwell from April 16th to 22d. She has been under bromides for the past six months, and these have reduced the number of attacks as stated.

C. L. DANA,
M. A. STARR.

The ocular examination made May 11th by Dr. Webster showed that she had: R. hyperphoria, 7° to 9° ; esophoria, 8° ; abduction, 5° ; myopia, (4.50 D.) Rt. and compound myopic astigmatism (8 D. = - 3.50 D. C. ax. 180°) left.

Oct 23.—She has had numerous operations during the summer; she feels better; she has had no severe convulsions since May 23, when she had one; she has slight seizures three or four times monthly.

Nov. 4 —She reports that she is now having only one or two attacks weekly, whereas before Dr. S. operated she was having five to six severe attacks a week. This latter statement is entirely inconsistent with the history as taken in April, and is therefore worthless. Her condition is therefore about the same as when she was under bromide treatment. But contrasted with her condition last fall under no treatment whatever, when she was having one or two attacks a day, she is very much improved.

Subsequent Clinical History.—This patient was steadily under Dr. Stevens' treatment up to time of present record, Nov. 4th, and during this time took no bromides. Two weeks after leaving off bromides, May 23d, she had a haut mal attack and another Sept. 15, 1889; she has had no others. The petit mal attacks have been reduced from one almost every day to one or two weekly. When seen in April she said she was having petit mal attacks two or three times a fortnight, (vide record); now she says she had them nearly every day. A letter from Dr. Webster says that when seen by him Nov. 4th she was having petit mal attacks once or twice weekly. Hence her stories disagree and the question of her improvement must remain an open one. She is certainly not cured.

Ocular History.—She has been operated upon nine times (Dr. Webster) and has worn correcting glasses for the myopia and astigmatism.

The following are records of these operations from notes made by Dr. Webster:

May 11.—R. hyperphoria, 7° to 9° ; exophoria, 8° ; abduction, 5° .

May 13.—Operation of tenotomy of right sup. rectus, leaving R. hyperphoria 3° .

May 31.—R. hyperphoria, 1° ; exophoria, 7° . Tenotomy of right internus, leaving exophoria, 3° , and no hyperphoria.

Her condition Nov. 4th, as reported by Dr. Webster, is:

R. V. $\begin{smallmatrix} 20 \\ - \end{smallmatrix}$ with 4 D.
 30

L. V. $\begin{smallmatrix} 20 \\ - \end{smallmatrix}$ with 9 D.
 70

Hyperphoria, 0° , without correcting glasses; exophoria, 2° .

With her myopia corrected, exophoria, 1° . Ophthalmoscope shows large staphyloma posticum both eyes, the left much larger and extending all around the disk.

No. 13.—Mary A. M., school-teacher; epilepsy; Dr. Dana.—The family history is good. The patient had no infantile convulsions or nervous diseases during childhood; when 13 or 14 years old she began to have attacks characterized by scintillations of light in the left eye, followed by blindness, but not by loss of consciousness and unaccompanied by twitchings or pain. They lasted one to three minutes. The present attacks came on at the age of 22, after unusual hard work. They came on at first at night, during sleep, and she had at first only three or four yearly. They came on after menstruation, but the menstrual influence was not absolute. About four years ago she began to have them in the day time and they increased in number until when seen by me in March, 1889, she was having two or three monthly. The attacks were preceded by the visual aura such as she had had for many years. They were accompanied by some convulsive movements and loss of consciousness, but were as a rule brief and abortive though occasionally severe. She had no *petit mal*. She had taken bromides a great deal since 1884, though not regularly. She complained of mental hebetude and loss of memory, though she was able to continue her school duties. She took bromides for but a short time under my direction, but was soon referred to the Commission for treatment.

Ocular examination by Dr. Moore, May 11, after an operation by Dr. Stevens, showed strabismus divergent ten degrees and strabismus convergent Rt. three degrees, myopia and myopic astigmatism. She went to Dr. Stevens March 28th and was first operated upon May 11th.

Subsequent clinical history up to Dec. 2, 1889:

The patient reports that she had

In March,	4	light seizures.	
" April,	8	"	
" May,	7	"	(2 severe).
" June,	9	"	(4 ").
" July,	18	"	(3 ").
" Aug.,		"	(1 ").
" Sept.,		"	Record incomplete.
" Oct.,	5	"	" "
" Nov.,	11	"	(1 ").

She states that her mind is clearer, she feels better, her attacks are lighter and not preceded by the aura; they are not lessened in number; she feels improved.

The records show very little improvement, as will be seen in the disease itself, although her subjective sensations are better. She still has the abortive attacks as often as ever and still has severe attacks.

OCULAR HISTORY.—The patient was operated upon five times. The records are given.

May 11.—Tenotomy of right externus with tendon resection of left externus, leaving exophoria five degrees. Same operation had been done on left eye a week before.

May 13.—The eye showed exophoria four to six degrees, with abduction twenty degrees.

Oct. 26.—Examined by Dr. Moore, who found: Abd. six degrees, add. twenty degrees, esophoria two degrees at twenty feet.

No. 14.—Agnes H., 31 years old Epilepsy, Dr. Birdsall.

Family history good, except an aunt possibly had epilepsy.

Has had scarlet fever and other diseases of childhood, otherwise healthy. Ten years ago had a severe epileptic attack. Three years ago (1885) after death of mother, was taken with light seizure, without aura, severe convulsions or subsequent somnolence. Since the increase in the frequency of the attacks, and also in their severity, interval never more than a month, often several slight attacks in a day. Has been subjected to various forms of treatment. Menstruation irregular, is anaemic and constipated. Acknowledges former habits of masturbation, which at one time was very excessive, but claims that she does not indulge in it at

present. Patient is easily excited and becomes tremulous under slight excitement. Sister describes attacks as follows:

Eyes are open, face is pale, does not usually cry out, except in severe attacks, then utters "an awful sound like a nanny goat's cry;" convulsive movement becomes general, sometimes has them while standing or sitting, usually they occur while in bed or sitting. Previous to date has not fallen but twice, does not bite tongue, but froths at mouth. Usually opisthotonus, left hand pressed against right leg, in convulsion, patient so rigid that they cannot move her. This stage of tonic and clonic spasms lasts about one minute then succeeds a period of confusion in which she often wanders about and does strange things, is dizzy and and sleeps at times; does not respond intelligently to questions at this time.

Subsequent Notes.—Patient was put under bromide, Dec. 27, 1888, and attacks were partly controlled, but symptoms of brominism occurred under doses that barely prevented attacks. The slight attacks occurred, as shown in record, were chiefly at times when bromides were reduced to correct brominism. Then during January, February and March, the patient had but one severe attack, (9 in January, 6 in February and 9 in March), and intervals of seven days occurred without any. A severe attack occurred March 30, and a series of 7 from April 2 to 5. At the time of series of seven attacks, in April, she had been under valerian alone. While free from bromide on April 20, another severe attack occurred. It was seen by Dr. Seguin, who confirmed diagnosis. She went to

CASE 14.—ANNUAL REGISTER OF ATTACKS.

Severe attacks, x.

Slight attacks, .

1889	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	X	.
Jan'y.																																	9
Feb'y.																																	6
March.																																1	9
April.																																8	15
May.																																2	29
June.																																3	19
July.																																	36
August.																																	24
Sept'r.																																	
Oct'r.																																	
Nov'r.																																	
Dec'r.																																	

see Dr. Stevens, April 25, and bromides were discontinued. Glasses were given the 27th. During May, patient had two severe attacks, and a greater number of light attacks than for any month previous since she came under observation. 29 light attacks, sometimes two a day, and once three. During June she had three severe attacks, and 18 slight attacks. During July, 30 slight attacks but no severe one. Twice she had three attacks a day and on one day four. On August 24, slight attacks but no severe ones. In September, one severe one and ten slight during the first eight days of the month. Then her own copy of chart of attacks was lost, but she says that attacks occurred nearly every day, sometimes twice a day, about as in the previous month. She was seen October 17, and reported seven attacks October 14, and one slight one each day after. Her general condition was about the same as on previous visits. She had been seen by Dr. Stevens and given glasses.

Record is herewith subjoined: February 6; March 12; April 15; May 29; June 20; July 30; August 4; September, attacks nearly every day, sometimes two; record destroyed; October 13 to 30, 21.

Ocular History.—May 13. V. $\frac{3}{80}$ in each eye not improved by glasses. Exophoria 6°. Abduction 14°. Tenotomy of the left Externus was done, leaving orthophoria and abduction 6°.

D WEBSTER.

October 30.—I examined A. H. this evening. She is an epileptic of 11 years standing, and she and her sister do not think she is much improved by the two operations that Dr. Stevens performed on her eyes. V. = $\frac{3}{80}$ + in each eye, but sees $\frac{2}{13}$ with both eyes at once with + 1 D. Accepts no glass with either eye singly. Hyperphoria 0°, Esophoria 1° to 2°. In accom. exophoria 9°, abduc. 5° adduct 27°. Sursumduction R 1° L 1°. Opthal. exam. short, pale temporal halves of discs with physiological excavation both. Visual fields roughly tested seem normal. No asthenopia and no diplopia or limitation of movement of the eyes.

D. WEBSTER.

TABULAR SUMMARY.

	CURED.	IMPROVED.	UNIMPROVED.	UNKNOWN.	TOTAL.
Epilepsy, . . .	0	3	5	1	9
Chorea,	0	3	2	5
	0	6	7	1	14

Correspondence.

A CORRECTION.

To the Editor of the Journal of Nervous and Mental Diseases :

The report of the discussion on the results of the work of the Stevens Commission, published in the JOURNAL of November, 1889, contains letters from Drs. Weir, Amidon, and Ranney regarding the condition of some of my patients. These gentlemen make statements which directly contradict my own report upon these cases. In the discussion I explained upon what facts my opinion was based; but your published report does not contain these explanations, and I ask you, therefore, to permit them now :

1. The case of Gertrude W. was put down by me as improved, and about her there is no controversy. She still has epilepsy both in *haut mal* and *petit mal* types.

2. Mary McK. I put down unimproved for the simple reason that, though she felt better, yet she was having just as many fits as when I first saw her, according to my records. A few weeks before the report was made, Miss McK. called at my office and made a statement of the above effect to me.

My notice of her case, in the clinic-book at the Post-Graduate Hospital, shows that, according to her statement in March, 1889, she was having two or three attacks of *petit mal*, or rather of abortive *haut mal*, monthly. The same statement was made to me at a second examination in April, 1889. Going by these records, and not by the *present* recollections of the patient, I concluded that her epileptic condition was essentially unchanged. This view is certainly being confirmed, for during the first week in December last she had two severe attacks and three light attacks.

3. In the case of Aggie H. my record, made December, 1887, was that she was *having then* two to four attacks daily, but that she had had six to ten daily. This record agrees with Dr. Fisher's, whose patient she was. The statement that she used to have thirty or forty attacks daily was not made to me, but may be true of her in past years. When sent to Dr. Stevens, however, she was having only the number stated, and she is having two or three daily now. It seems to me that her condition is not essentially changed, therefore.

4. The case of Patrick H. was reported slightly improved. He was not placed on the list of cases, because, as I understood, Dr. Stevens did not wish it, for his treatment was not kept up long enough.

Dr. Stevens' statements regarding him, however, are very inaccurate. He had had, before he was under the Commission's treatment, at the most five *haut mal* and many *petit mal* attacks weekly. The month before treatment, however, he was having only two or three a week, as my records show.

On December 5, 1888, he reported to me that he had had five *haut mal* attacks weekly and one to two *petit mal* daily. The improvement was very slight, and it seemed to be mainly in subjective sensations.

The case of L. G. (p. 711), cited as an evidence of unfairness toward Dr. Stevens, was fully explained by me in the discussion. The girl lived in a New Jersey town some twenty miles away. The family was poor, and could not bring her in for treatment. I found that it was impossible to make her a Commission case, much to my regret. She came to the Post-Graduate clinic a few times irregularly; and so I sent her to Dr. Moore for relief of her strabismus, which was very manifest. He corrected it; but I heard a little later that the child was no better.

Dr. Stevens' charge of unfairness is utterly unfounded. I sent twelve cases of epilepsy to him in good faith, all as suitable for treatment as I could possibly get. A few seem a little improved; but even admitting all that Dr. Stevens asserts about them, none are cured, nor do they in any wise approximate to a cure. I have, for my part, felt much disappointment in the results, for I had hoped much more from the method. As the evidence stands, so far as epilepsy is concerned, it seems to me that treatment of ocular insufficiencies is perhaps not always valueless, but is entirely inefficacious as a means of cure.

My relations with Dr. Stevens have always been agreeable, and I esteem him so highly that his charge of gross unfairness was something of a surprise to me. I will grant him improvement in all the cases he asks; but even then the "improved" cases are so far from being cured that the concession would not change my opinion of the efficacy of his method.

Very truly yours,

C. L. DANA, M. D.

50 W. 46th St., Jan. 2, 1890.

THE COLONIZATION OF EPILEPTICS.¹

BY FREDERICK PETERSON, M.D.,

Attending Physician to the New York Hospital for Nervous and Epileptic; Lecturer on Nervous and Mental Diseases at the New York Polyclinic and Vanderbilt Clinic.

IN 1887 I wrote an account of a visit I had a short time previously paid to the Bethel Epileptic Colony at Bielefeld near Hanover in Germany;² and I have long desired to give a more comprehensive description of that institution, and to agitate the establishment in this country of a somewhat similar charity under the government of a State or at the instigation of private philanthropy. For every other defective class provision has been made in greater or less degree, for the insane, for idiots, for the deaf and dumb and blind, for the sick and the crippled, for the aged and infirm, for young malfactors in reformatories, for the negro and for the Indian—and for all of these, improvement in care, treatment or development is continually progressing.

Almost alone, however, has the sufferer from epilepsy been left to shift for himself, usually an outcast from his family, expelled from the schools, denied industrial employment, shunned to a great extent by his fellows, left to grow up in idleness and ignorance, friendless, a prey to one of the most dreadful and most hopeless of human ills, refused admission to general hospitals, generally drifting at last to the almshouse and insane asylum. He is given refuge in the insane asylum, not as a rule because he is deprived of reason, although many epileptics do become demented or unmanageable, but because there is no other place for him to go. Hundreds, nay thousands, of epileptics are in American asylums at this moment, who do not really

¹ Read before the Section on Public Health, etc., of the New York Academy of Medicine, Dec. 6, 1889.

² New York Medical Record, April 23, 1887.

belong there, and of whom many will be found who show not even the slightest mental aberration. It is an injustice to the epileptic, and it is a detriment to the insane.

When we take into consideration that a large proportion of these unfortunates are gifted with as much intelligence as ordinary human beings, that they are as capable of education, as well adapted for industrial pursuits, quite as able to be self-supporting as most people, the unutterable woes of this class become more apparent.

The conditions under which they may secure their proper mental development and their meed of occupation, must be such as combine medical supervision with wise industrial teaching and training. Society has here to perform a sadly neglected duty.

Actuated by such considerations some twenty-four years ago, a Lutheran clergyman, Pastor von Bodelschwingh, purchased a farm in the suburbs of Bielefeld, and with four epileptics as a beginning, established a colony, which for nobility of conception and excellence of results is unique in our civilization.

It seemed to this benignant man that it was feasible to create a refuge where these sufferers might be cured, if curable; where their disease might be ameliorated, their intellectual decay prevented; where they might have a comfortable home if recovery were impossible; where they might develop their mental faculties to the utmost; might acquire trades, or engage in whatever occupation they cared to choose; finally growing into a community of educated, useful, industrious, prosperous and contented citizens. From that small beginning there has been a gradual evolution and expansion of the colony. In 1878 it had 250 epileptics; in 1880, 458; in 1882, 556; in 1886, 830; and at the time of this writing considerably over 1,000.

During the twenty years from 1867 to 1887, 2,407 epileptics had been received and treated at the colony. Of these 156 or 6½ per cent. were discharged recovered, and over 450 improved.

But the dispensation of good by the colony is not limited to its immediate inhabitants, for upon application, with

or without payment, medicines are sent to epileptics in every part of the world, without regard to sect or nativity, and the enormous extent of this export of remedies may be appreciated from the fact that 800 pounds of potassic bromide are now consumed per month by residents and correspondents. Some 48,000 epileptics have been beneficiaries of this charity.

EDUCATION AND EMPLOYMENT.—The industrial pursuits at Bielefeld are numerous and varied. Occupation not only ameliorates the mental and physical condition of epileptics, but the incurably afflicted are offered opportunities for culture and callings not attainable anywhere except in institutions of this character.

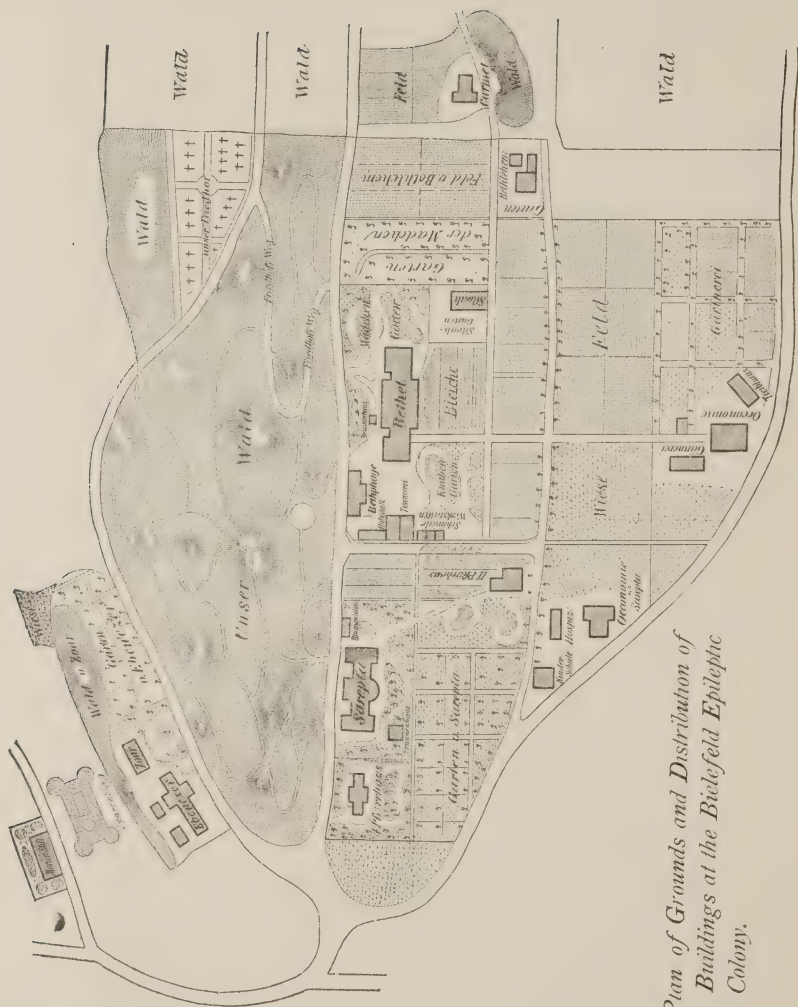
The Bielefeld Colony now consists of 55 houses, 150 families and over 2,000 inhabitants, inclusive of teachers, nurses, industrial trainers, co-laborers and patients. The arrangement of these houses can be seen in the accompanying plan. It will be observed that the colony has little or nothing about it to suggest an institution, but is rather similar to a country village, with its variegated cottages and pretty gardens and farms. The houses are scattered over 320 acres of beautiful woodland and meadow. A synopsis of the uses of most of the buildings is given below :

Female Division.

- 2 cottages for patients of the better class.
- 1 house for feeble-minded women and girls.
- 1 house for half-demented girls.
- 1 house for convalescent girls.
- 14 houses for school children and workers.

Male Division.

- 2 houses for patients of the better class.
- 1 house with 3 schoolrooms for boys and youths.
- 9 houses for hand-workers.
- 4 houses for gardeners and farmers, with gardens, farms and brick-kiln.
- 5 houses for more or less demented patients, 2 of which are for those still able to work.



One of the chief features of the colony is the system of decentralization, the division of the patients as much as possible into small families, occupying cottages, the separation of the sexes, of the feeble-minded from those with normal mental faculties, separation according to age, social standing, former occupations, etc.,—a system likewise applicable, I may say, to the insane in the ideal psychopathic hospitals of the future, and already to some extent applied in the Saxon institution Alt-Scherbitz, and in the new *Manicomio di Roma*.

For the purpose of securing a sufficient number of male and female nurses, and of a superior order of kindly and sympathetic care, it was deemed expedient to found at the Bielefeld Colony also nurses' training schools, and as a result of this, not only have they well-trained brothers and sisters forming an order of deacons and deaconesses for their own use, but they have been enabled to supply various hospitals and insane asylums with a high class of caretakers. For my own part, and from my experience in institutions, I shall always feel that the highest order of care and nursing for such unfortunates as the insane and epileptics can only be obtained by the employment of those who enter upon these most arduous of duties from a deeply religious motive.

To observe more closely the variety of employments in vogue, one must make a personal visit to this remarkable colony, which I did in the Winter of 1886-'87.

We find here schools in which instruction is given in all the branches usually taught in ordinary public schools; and to those who desire these, opportunities for higher studies in languages, arts and sciences are granted, many of the richer patients even employing private tutors for this purpose.

In the sewing-room, singing or being read to, may be seen sixty or more young women, making and repairing clothing, knitting stockings, doing useful fancy work, manufacturing wearing apparel and bed clothes.

In the kitchen and in the vegetable garden they may be seen busy with the preparation of meals, and the proper

rearing of garden produce. The bedrooms, dormitories and dining-rooms, the laundries, drying and ironing-rooms, furnish occupation for a very large number of other women.

The men naturally have greater variety of employment. There are the book store, printing establishment and book bindery; a room for those who delight in illuminating picture-cards and mottoes for mural decoration, a favorite art everywhere in Germany; at the bindery, pictures are framed, books bound, envelopes made, and some leather work done; the books printed are in particular popular works for the dissemination of moral and religious instruction; twenty men are employed at this work. Floriculture, agriculture and fruit-raising require large numbers of employees, particularly so, since so much flower and vegetable seed-produce is garnered, put in packages and shipped and sold from their own seed-store.

A joinery, bakery, tailor-shop, paint-shop, locksmith-shop, blacksmith-shop, foundry, tin-shop, shoe-shop, saddlery, dairy, brick-yard, drug-store and grocery, all provide occupation to numberless inhabitants of this colony, even to some suffering also from old hemiplegias.

Some of the houses have been planned by epileptic architects, the brick made by sixty epileptic patients at the brick-kiln, the masonry done by epileptic workmen, the woodwork made by their own carpenters, the ironwork by their own smiths, the painting, glazing, furnishing by their own adepts in these various trades.

For men alone there are over thirty different callings.

From this it will be seen how noble was the conception of this truly philanthropic institution. And not only in manifold employment has the genius of its conceiver been made manifest, but in all things that might tend to distract the minds of the patients from themselves and their misfortunes. Games and amusements are many, walks in the gardens, out-of-door sports, evening entertainments, singing schools, an orchestra made up from their own number, a museum for the collection of stamps, coins, gems, autographs, bronzes, antiquities, articles of ethnographic or historical interest, and specimens from the animal, vege-

table and mineral kingdoms—all have been carefully thought out for the perfect evolution of this little social world.

When the evil disorder attacks one in the field or garden, willing hands are near to attend to him, and every workshop has its mattress within convenient reach on occasions of emergency.

To sum up the objects of this colony, it is in the first place a hospital for the cure of epilepsy, and attempts at cure are made upon every patient without exception. It is a school for the instruction and education of epileptic children. It is an industrial institute for epileptic youths and adults. It is an asylum for those who become demented from the effects of the disease.

Although the colony is under the domination of the church, and has a Lutheran pastor at its head (I do not know that a physician or ordinary layman with his more sordid purposes would be as efficient a superintendent), three physicians are employed to supervise the medicinal treatment, one of whom is resident in the colony.

TREATMENT OF EPILEPSY.—It is to be expected that something should have been learned of the best methods of treating this disease after an experience of so many years with such immense numbers of patients. As a result of the experience of the colony, the following is the course now pursued :

1. *Employment of mind and body.*—This is paramount. Out-door work is best. It has already been shown how admirable is this therapeutic feature in the Bielefeld colony.

2. *Restrictions in diet.*—Food must be plain, not rich, and little acid. Spirituous liquors are to be avoided. (Smoking is allowed in moderation.) Coffee and tea must be weak and mixed with much milk. A good milk diet is preferable to meats or soups. Supper should be early and light.

3. *Baths.*—Cold shower baths gradually prolonged, and cold sponge baths are beneficial.

4. *Drugs.*—Everything recommended in the pharmacopeia has been tried as to its efficiency in this disease, and

nothing found to succeed so well as the bromide of potassium alone. All of the bromide salts have been experimented with both singly and in combination, and though the mixed bromides are often of great value, the potassic salt has in every respect proved to be the most satisfactory. It has been found that most patients can take it year after year without gastric disturbance. It cures some cases, lessens the frequency of seizures in most, soothes the excited nerves, and serves to diminish the tendency to mental enfeeblement. The usual prescription is

R	Pot. brom.,	-	-	-	20 grammes, (3 v.)
	Aquæ puræ,	-	-	-	200 " (3 vi.)

SIG.—Take one tablespoonful three times daily, immediately after eating, for the first week.

If the seizures diminish in frequency, keep to the same dose; if not, increase to four tablespoonfuls daily *in the second week*, five *in the third week*, and so on until eight such doses are taken daily if necessary. If eight prove injurious mentally or cause severe eruptions, particularly on the legs, the dose is again gradually diminished. In many cases minimal doses only are borne and should then be persisted in for indefinite lengths of time. The above dosage is for patients over sixteen years of age. From ten to sixteen, begin with three tablespoonfuls, but do not exceed five or six. Under ten years begin with two such doses daily and increase to four. Very young children should be given still less.

Particular pains should be taken to procure a thoroughly pure drug, as the bromide generally sold contains impurities, such as chlorate of potash and other foreign substances, often to the extent of six per cent. An especially pure drug is prepared for this colony, containing not above 0.5 to 0.7 per cent. of foreign matter.

Even after the cessation of attacks the remedy should be persisted in with gradually lessening doses, for as long as eight to nine months.

OTHER COLONIES FOR EPILEPTICS.—Sine the successful establishment of the Bielefeld colony, a number of

similar institutions for epileptics have sprung into existence on the Continent, particularly in the various provinces of the German Empire. Among these are Rotenburg also in Hanover.

Mariahilf near Munster and Olpe in Westphalia.

Alexianer-Kloster at Aix-la-Chapelle and Rath near Düsseldorf for the Rhinish province.

Neinstedt-Thale for Saxony.

Tabor near Stettin for Pomerania and Posen.

Karlshof near Rastenburg for East and West Prussia.

Potsdam for Brandenburg.

Haarlem in Holland.

Zürich in Switzerland.

Thus the beneficent work is extending in many directions, and surely it is time that philanthropic minds should in this country also turn their attention to one of the most unhappy classes of our people, a class more deserving of commiseration, more deserving of the title of "the children of the State" than many vicious classes whom we endeavor, usually vainly, to reform in large government institutions.

At the usually accepted ratio of two epileptics per thousand of population, the United States have much over one hundred thousand persons suffering from this disorder, the circumstances and conditions of most of whom are such as have been described. There is probably not an almshouse or asylum in any county of this Union but has a few or many epileptics among its inmates. As far as I know there is no institution in this country existing especially for this class, save the small beginning recently made by the State of Massachusetts in converting a private asylum for epileptics into a public one subsidized by the Legislature. The New York City Hospital for nervous and epileptic, on Blackwell's Island, of which I am one of the attending physicians, is hardly to be called a hospital for this class alone.

In New York State there are some 12,000 epileptics. The poorer classes have drifted to a large extent into public institutions, chiefly almshouses. In the more important State asylums there are now about 300. Utica has 21,

Binghamton 80, Middletown 19, Willard 160, and Buffalo 22. They are far from desirable patients to associate with the insane, and are taken into these institutions only because there is no other place for them. They are received under a sort of compulsion.

Dr. J. B. Andrews, superintendent of the Buffalo State Asylum, writes me: "I should prefer a separate institution, especially for the younger class of epileptics."

Dr. G. Alder Blumer, superintendent of the Utica State Asylum, writes me: "In my opinion there should be a special State custodial institution for epileptics."

Dr. T. S. Armstrong, superintendent of the Binghamton State Asylum, writes me: "We have thought there should be a separate institution for this class. We find epileptics often have a depressing influence upon our other patients."

Dr. S. H. Talcott, superintendent of the Middletown State Asylum, writes me: "Epileptics should not, in my judgement, be admitted to general asylums for the insane. Hospitals should be constructed especially for their needs."

New York City which is largely representative of every thing in the Union, is also representative of what the Republic has done, or rather has failed to do, to mitigate the misfortunes from which this class suffer more than any other. In the first place a great proportion of epileptics are cared for in their own homes, wretched as these may be, and seek in large numbers the consolation of treatment at our city dispensaries. I know personally of perhaps a hundred who are or have been regular attendants in the Nervous Departments of the Vanderbilt Clinics and Poly-clinic within the past year.

When even this doubtful solace of idly living at home, without education or employment, and of frequenting the dispensaries, cannot be continued, the City reluctantly extends to them its hospitality—the hopeless comfort of its almshouse and its numerous asylums. There are large numbers of epileptics in the almshouse on Blackwell's Island. My wards in the Nervous Hospital contain some fifty within its cheerless walls. There are large numbers in the Women's Insane Asylum on Blackwell's Island, in the

Men's Insane Asylum on Ward's Island, and other of the City's capacious charities open to them some sort of refuge from the world's calamities and distress. But the utter inadequacy of such institutions to the needs of the epileptic is too apparent to require further word of mine.

This essay has been written in the hope that some public sentiment may be aroused as to the necessities of this neglected class of defectives, in the hope that it may prevail upon some religious sisterhood, some private philanthropist or some public official to provide for the early establishment in this or some other State of an epileptic colony, which shall become a home for the homeless, a place of refuge from many miseries, an educational institute for those who are forbidden the public schools, an industrial college for those to whom the ordinary avenues of trade are closed, a hospital where cure or palliation shall be possible, and where the highest scientific minds may be enabled to discover sometime a specific against one of the most woful of human ills ; in short, a prosperous, industrious and thriving community, to serve as a model for many other such yet to be founded on this continent.

201 West 54th Street.

INSTITUTION CARE FOR THE FEEBLE-MINDED.

By SAML. J. FORT, Md.,

Superintendent of the Fort Hill Private Institution for Feeble-Minded and Epileptic Children,
Ellicott City, M. D.

IT is one of the most promising indications of the growing insight into what humanity owes itself, that amid the din and roar of the battle of life, the advancing army of civilization finds time to render assistance to the non-combatants; those unfortunates who, handicapped by defect and crippled by infirmities, would be crushed under the wheels of progress, or fall a prey to the mauling hosts ever prowling upon the outskirts, without a helping hand. The insane, the deaf, the dumb, the blind, have received special care for several generations; under specific training the unimpaired faculties are made to counter-balance those lost, and comfortable homes for the indigent of these classes have been established. Yet one great class—certainly the most helpless, possibly the most suffering—was overlooked and almost forgotten, until a few decades past. The feeble-minded, who have seeing eyes yet see not, hearing ears yet hear not—innocent sufferers for sins they did not commit, sitting in the shadow of a helplessness, infinitely greater than that of other defectives,—these most afflicted of the afflicted,—found all doors shut against them save those of the jail and the poor-house.

The existence of this class was acknowledged by the ancient writers. Thus, Vitruvius mentions the cretins and their goitres as monstrosities, and up to the beginning of this century idiocy was regarded as the result of supernatural influences.

Martin Luther claimed that idiots were possessed with devils, and in one instance urged the drowning of a particularly hideous cretin in the Moldau. As a consequence of these views, he denounced people who should attempt to alleviate the affliction or cure it as "blockheads."

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The medieval literature of Germany and Scandinavia abounds in stories of changelings, or the offspring of hobgoblins, left by these supposed beings in place of the babe, which they snatched from the cradle and carried to their subterranean abodes. The description of these changelings, conforming so closely with that of cretins, these fables seem to indicate that at least in cases where the defect was not apparent at birth to untrained eyes, it was ascribed to an interference of occult and evil powers. Other nations, the Irish for instance, held the "innocents" in reverential awe as marked by God's finger. The Hindoos, while not considering idiots possessed with devils, shun them, or keep them closely confined, lest by some overt act or contact they cause a loss of caste, the most serious calamity which can befall the followers of Brahma. In Mahometanism we find a rational understanding of the question. The Koran commands that if a man has a feeble-minded brother, he give him not his part of an inheritance to hold, but give him clothes and sustenance therefrom, and speak kindly to him.

At the dawn of this century not a page in scientific, philanthropic or religious literature had been devoted to idiocy beyond comments upon endemic cretinism, in certain valleys of Switzerland and Savoy, except a few religious establishments in the mountains of Switzerland, which sheltered a limited number of cretins, no effort had been made to care for such unfortunates, and, until Itard's experimental training of the so-called "wild man of Aveyron," nothing had ever been done to ameliorate or improve the condition, either mental or physical, of the feeble-minded.

Stimulated by the results of Itard's experiments upon this neglected waif of humanity, Edouard Seguin, a student under Itard, began the training of idiots by methods of his own, and making close studies of their psychological conditions, produced very remarkable results, and his successes being published, attracted universal attention. Continuing his work in Paris with unabated interest, Seguin was complimented by the Parisian Academy of Sciences, which

publicly declared through its committee, that he had solved the problem of successfully training the idiot. Dr. Guggenbühl, a young physician of Switzerland, a few years later founded his institution for the treatment of cretins in the Abendberg near Interlaken, in Switzerland, which, while it proved a partial failure, served to further increase interest in the work, and from Paris and Switzerland as centres the great wave of charity for these poor creatures spread all through Europe and crossed the ocean into America, where, in 1848, the late Dr. Hervey B. Wilbur opened a school at his home in Barre, Massachusetts, from which beginning sprung the institutions of this country, caring for a grand total of over 5,000 feeble-minded.

The treatment of the idiot and the task of developing to the utmost such abilities as are latent in him, is not alone confined to the therapist. He is a subject for pedagogic as well as medical treatment; he is a pupil as well as patient.

It is perhaps a common error to assume that the idiot can be "cured," in other words, that man can make normal what nature has made abnormal, or, on the other hand, to assume that nothing can be done for him save to make him comfortable and keep him out of harm. Serious and momentous indeed is the question, "what can be done for the idiot?" to every heart that throbs with human sympathy, and it comes with much sadder emphasis from the parents of an afflicted child. Insanity is a disease, idiocy a defect or abnormal development. It has been aptly said that the insane man is like one who has been rich, and lost his wealth, the idiot like one born in poverty. A disease may be cured, but human instrumentality can never remodel nature's incomplete products and constructive failures; it can only do the best possible with the material given, and the feeble-minded must remain so even under the most complete training. This fact established, what does remain?

Let those who might think that when we cannot reach the impossible, the possible is not worth struggling to attain; not worth the life-work of noble men and women,

or the full support of taxpayer or philanthropist; let them visit an institution for the feeble-minded; let them look at a child brought by a mother, whose careworn features bear witness to the fearful infliction the care of an idiot is to a mother. The child, fretful, noisy, restless, of filthy habits, making home a place of never-ceasing torment to itself and its associates; unable or unwilling to work; its hands useless appendages, or at most, means of senseless destruction to objects within reach; through its very affliction working a despotism over those who love it, to its own detriment. Let them watch the gradual development of this child under the institution training; see it taught to conform to orderly habits of life, to cease causeless noises, to enjoy and exercise cleanliness and order, to take pride and pleasure in self-help; the idle hands gradually beginning to obey rational impulses, and ceasing to destroy as they learn to create, however humble the result may be; it will then be seen, as is daily seen by those who are working with such children, the joyful pride with which the first little piece of work completed is shown to everybody around them, and the importance of the words, "I made that," duly appreciated.

"But," some will say, "the same results might be attained at home, what, therefore, is the necessity of an institution?" Theory and experience are here hand in hand, both proving that only in rare instances has home talent the ability to bring out the results of the institution; and a German authority says, that where this may be found time nor opportunity would be obtainable to devote to the work without doing injustice to the rest of the family. The education and training of the feeble-minded is a specialty learned only by study and long practice.

Under ordinary circumstances the home environments are the most unpropitious for the welfare of the feeble-minded. He is at all times the skeleton in the closet; at social gatherings he is put aside that his presence may not mar harmony. He is debarred the privileges of the church and Sunday-school for the same reasons. Normal children of his age shrink for him, or taunt him with his condition,

hence his home life is more or less confined, the isolation thus compelled aggravating and not ameliorating his condition. The dietetic treatment, the importance of which in the general care cannot be overestimated, must of a necessity be disregarded at home; and last, but not least, the presence of a defective in a family is always a shadow—a burden, however lovingly assumed and conscientiously borne; it taxes the mother's strength beyond endurance, robs the other children, if there are others, of their proper share of attention, and among a certain class the poor imbecile is treated cruelly, subjected to every indignity, hardship and privation, because he is a useless consumer, not able to earn his pittance in the mill or on the street.

The institution furnishes a little world, a microcosm, with its curriculum of duties and pleasures. The feeble-minded child finds company, his isolation ceases, he is no more hidden away when there are visitors; he is placed in a school with other children, among whom he finds congenial playmates; he is placed under the guidance of a teacher who knows how to call forth all his slumbering faculties, at the same time he almost unconsciously comes under dispassionate, firm, yet gentle discipline. There is no sphere of charity more deserving the attention of those who have enough and to spare and are philanthropic enough to devote part of their surplus to promote the interests of stricken humanity. That the community has moral duties towards its idiotic population is no longer questioned. The history and etiology of idiocy points out the necessity of sequestering the idiot and the imbecile in special institutions for the following reasons:

To remove the incubus of idiocy from the house of the people which it blights.

2. To prevent idiocy from begetting idiocy, which is much too frequent among imbeciles in almshouses, where the association of the sexes is not properly restricted.

3. To educate and train them as far as possible.

4. To alleviate the complication of the affliction, through proper dietetic and medical cure, and thus prevent the less afflicted from degenerating into profound idiocy.

5. To make available the industrious abilities possessed by a large percentage, which experience proves utterly unavailable outside of an institution.

6. To train the less afflicted to take care of the very dependent under proper guidance.

7. To save them from evil and temptation which they are unable to resist, by placing them in a miniature world *sans* its temptations.

It is a sad fact that among those imbeciles who most nearly approach the line of average intelligence, decided criminal proclivities are found, and many unquestionable imbeciles are inmates of our penitentiaries. That female imbeciles will fall victims to the first unscrupulous scoundrel who takes advantage of her feebleness, needs no further elaboration, and this reason alone should demand the utmost sympathy from philanthropy, aside from the moral obligations of the strong to protect the weak, and the utilitarian policy of prevention in the bud rather than cure in the full-blown flower.

PERISCOPE.

By GEORGE W. JACOBY, M. D., L. FISKE-BRYSON, M. D. AND
F. H. PRITCHARD, M. D.

TREPHINING FOR CEREBRAL HÆMORRHAGE: A SERIES OF
TREPHININGS FOR VARIOUS OCCURRENCES, VERTIGO,
TRUE AND SYMPTOMATIC EPILEPSY.

Championnière (*Journal de Médecine*, p. 532, 1889) presented to the Académie de Médecine, under the above title, a fresh case and statistics of thirty cases of operation, without a single case of death or untoward occurrence. These thirty operations were performed in cases in which there had been no antecedent traumatism. The new case, here added, is as follows:

A man, aged fifty-three years, had an attack of cerebral hæmorrhage twenty months ago. Right hemiplegia; late contracture of the hand and epileptiform attacks ensued. Focus was localized at the middle part of the precentral convolution. By means of craniometrical measurements the corresponding portion of the skull was mapped out, and the trephine applied. A focus of an old cerebral hæmorrhage was found. The focus was removed and the surrounding walls carefully cleansed with antiseptic solutions. Suture of dura and scalp; drainage. Duration of operation one hour and a quarter.

The day following the operation the contracture of the hand had ceased. Power had also greatly returned. When the patient was allowed to get up, his walk was found to be easier. His speech is more distinct and his intelligence greater. During four months he has had no convulsion, whereas prior to the operation they recurred at least every two weeks.

UNE FAMILLE DE DÉGÉNÉRÉS INCENDIAIRES.

Dr. Émile Laurent (*Annales Médico-Psychologiques*, p. 355, 1889) states that pyromania may develop under three

distinct forms. In some cases it is simply an episode in the course of another more or less systematized delirium. In other cases it is the result of an irresistible impulse. This is real pyromania; here the pyromaniac burns, the same as the dipsomaniac drinks. Finally, the third class of pyromaniacs are those individuals who are suffering from some form of degeneration, and who commit the incendiarism as a result of some futile motive.

He gives here the history of an entire family of pyromaniacs. All the members of this family are hereditarily degenerated, almost weak-minded, without will-power or constancy. In this family there are three incendiaries. These three differed from ordinary pyromaniacs, inasmuch as the incendiary impulse did not develop at the time of puberty; the one case had long passed this period, and the two others had not yet reached it. Neither did they seem to obey any morbid impulse returning periodically and under almost identical circumstances. On the contrary, they were always guided by a certain motive. In the one case it was the desire for vengeance, any supposed insult being avenged by throwing a burning torch into the enemy's house; and the other two it was simply self-gratification, the pleasure experienced at seeing the flames.

These cases differ legally from the cases of incendiarism due to morbid impulse, for the former suffer only from a weakened will, and are able to combat the criminal idea, while for the latter any struggle is impossible.

UN CAS DE PSYCHOSE ÉPILEPTIQUE.

Dr. Chatelan (*Annales Médico-Psychologiques*, p. 383, 1889) first recalls some well-known facts about psychoses following epilepsy, and then gives the detailed account of a legal case. The case is instructive in so far as the patient apparently was the possessor of perfect intellectual health. A young man occupies himself with all the duties required of him, does office-work, studies at a commercial college, and leads his daily life without exciting any surmise that he is not as he should be. Suddenly, without any provocation, he makes an attack upon a stranger, is arrested, and medi-

cal examination shows him to be affected with petit mal. C. finally says: "Any unaccustomed criminal act, absurd in its ends or means, must, even if the person committing it is apparently in the most perfect health, be made the object of a careful medical examination." G. W. J.

RACHITIC PSEUDO-PARAPLEGIA.

In the *Medical Record* of Nov. 16, 1889, Dr. Henry W. Berg makes some statements of value and interest concerning this condition, of which the symptomatology is about as follows: The little patient, of two, four, or even five years of age, is unable to walk, and younger children to stand or sit up. Efforts to walk are not made without aid, as a general thing; but when such efforts are made, the feet and legs are spread wide apart—for better basis of support—and the body bent forward to maintain equilibrium. Stumbles and falls are frequent, and the little patient walks like a partial paraplegic. Examination shows a rachitic body; costal cartilages marked by so-called rachitic rosary; tenderness in the body of the muscles and over bony prominences at muscular insertions. Demonstration proves that every muscle retain its power intact.

Rachitis is not a disease of the bones, *per se*, but a disease of the blood, affecting all the structures of the body, with lesions especially located in osseous structures, though not confined to these, but affecting materially muscles, ligament, and fibrous tendons. Differential diagnosis in forms of paraplegia seen in childhood is sometimes difficult. Electricity will settle the point in regard to poliomyelitis and rachitis. In the first condition, the muscles do not respond to the faradic current. In the second, contractility is preserved and normal. The reaction of degeneration is absent in rickets, the reaction being normal, $\text{Ca.C.C.} > \text{An.C.C.}$, $\text{Ca.O.C.} < \text{An.O.C.}$, or entirely absent.

Paralysis due to poliomyelitis has the characteristic galvanic reaction of degeneration. It is with greater difficulty that paralysis following diphtheria and cerebro-spinal meningitis is distinguished from rachitic pseudo-paraplegia. In these the nerve-centre lesion being temporary, recovery

is the rule. Atrophic appearances and reactions of degeneration are absent. Mild cases of spastic or tetanoid paralysis present increased patellary reflex, exalted muscular activity, and retarded mental development. Paraplegia occurring in Pott's disease may come on slowly and be only partial. Careful examination reveals disease of the spinal vertebræ, with characteristic symptoms. To understand the pathogenetic connection between the rachitic disease and this pseudo-paralysis, the mechanics of muscular activity must be considered. When contraction of its muscles sets a limb in motion and makes it perform work, the sum total of the work performed consists of (1) counteracting the weight of the limb to be moved, and (2) imparting to the limb or member the force necessary to perform activity. The sum of the two is the amount of muscular work. When standing quietly, the entire work of the muscle is spent in maintaining the stiffness of the joints and counteracting their tendency to yield under the superincumbent weight of the body and limbs. In walking, more work is performed, the body being supported and carried along in spite of friction and other impediments. Muscular tissue, weakened by imperfect nutrition of rachitic blood, is sometimes unable to perform both of these tasks, the utmost force exerted only sufficing to maintain the body erect, though when the weight of the body is supported the movements of walking are frequently well and evenly performed. This explains why the patients move about nimbly in every possible direction when lying down, as they then support only the weight of the limbs, and not that of the whole body. This is also an explanation of the easy use of the upper extremities. When, however, these same little patients attempt to lift weights, they fail most lamentably, owing to increased flaccidity of the ligaments, softness of the bones (levers by which the muscles act), and the rachitic periostitis present, especially at the points of muscular insertion. To this periostitis is probably due much of the thickening and tenderness at these points. Pain is also an important element in the disability in these cases. When every motion causes pain, a child instinctively remains quiet.

Prognosis is always favorable. Treatment should be directed locally to the weakened muscles, and generally to the system at large, to counteract the effect of rachitic malnutrition. The latter indication is more important than the former. Removal or improvement of the fault in nutrition gives the muscles strength to perform physiological work. Diet is of the utmost importance. Whatever can be most easily digested by the patient, without regard to its richness in phosphates, is the proper diet. Milk (no tea, no coffee), thin cocoa, lean meat once a day, beef-broth in abundance, all of the lighter cereals and small quantities of the lighter fruits, cod-liver oil in small doses (not with hypophosphites), form an excellent anti-rachitic diet, remembering always that whatever disagrees must be instantly eliminated and something found to take its place. Other agents are massage, electricity, phosphorus and iron in alternation, small doses of nux vomica, etc., when anæmia is present. Satisfactory results are obtained from the use of Thompson's Solution of Phosphorus, according to the following formula :

R	Phosphori,	-	-	-	-	-	-	gr. j.
	Alcohol. absolut.,	-	-	-	-	-	-	℥ cccl.
	Spt. menth. pip.,	-	-	-	-	-	-	℥ x.
	Glycerinæ,	-	-	-	-	-	-	℥ ij.

M. and sig.

Of this mixture, from which the odor of phosphorus is entirely absent, to a child two to four years of age, six minims t. i. d., may be given, increasing the dose one drop weekly until ten drops are given. Correspondingly smaller doses are best for younger children. Larger doses for any are unnecessary.

L. F. B.

MANIA OF PUBERTY.

(By *M. Mairêt*, "*Ann. méd. psych.*," 1889.)

In addition to the consideration of a case (a girl, nineteen years of age) which was treated successfully, Mairêt considers the different opinions which have existed heretofore on this form of mania. He refers to the well-known

works of Griesinger, Morel, Kahlbaum, Hecker, Maudsley, and others, and on the basis of his own observations he comes to the conclusion that the process of development of puberty has in itself a great pathogenic importance toward the development of this mental disturbance. The process may take a normal, physiological course or a severe, pathological one. In the latter case the process demonstrates itself by bodily weakness, irregularities of the menses, chloro-anæmia, headache, hysterical attacks, change in the character (in the female); by a feeling of tiredness, headache, palpitation, cerebral excitement, etc. (in the male). The mania has a specific character, and Mairét distinguishes two groups :

1. Mania of puberty, with temporary cessation of mental development.

2. Simple mania of puberty, viz.: *a*, lypemanic stupor; *b*, choreatic mania; *c*, impulsive mania; *d*, hysteric mania.

In the lectures which have been published he treats of the *a* and *b* divisions of the second group. By lypemanic stupor he understands the “*melancholia cum stupore*”—Bailarger’s or the German expression, “*Katatony*” (katatonic). Of the seven cases cited, six were girls; three times puberty alone was the cause, in the four others there existed, in addition to it, a hereditary disposition. After a prodromal stage, which is marked by the symptoms of the difficult puberty, there follows, upon an opportune inducement, often after mental excitement, nearly always the sudden outbreak of the disturbance. In the first phase there exist anxious excitement, terrifying deceptions of the senses, especially of the sight, and sometimes fever, which then oscillates between 38° — 40° , may show an inverse type, and the objective examination reveals no change of the organs. In place of the depressive delirium, there appeared once religious exaltation. The stage of excitement leads, after a differently long duration, usually a duration of several weeks, into the stuporous one, in which, at the beginning of it, the hallucinations can be demonstrated; later on the latter disappear. The patients are unclean, helpless, salivate, cataleptic conditions appear, but the contractions of

the muscles are lacking ; the circulation is slower, the temperature decreased. Characteristic is the interposition of often, sudden, and transient conditions of the most vivacious excitement of one-quarter to one hour's duration, which distinguish themselves by impulsive actions, attacks of violence, grotesque dancing, monotonous repetition of the same words and sentences and the like, by which the stupor of puberty is differentiated from the stupor of other ages. The stupor lasts for several weeks or even months, and passes then gradually on, under increase of nutrition, clearing up of the senses, appearance of the menses, etc., to recovery ; sometimes the recovery is preceded by a stage of exaltation of short duration. The prognosis is in most cases favorable.

The *choreatic mania* appears more frequently in the male sex, and heredity plays a great part in this form of mania. Five cases. The prodromi are the symptoms of the difficult puberty, especially which are noticed long-lasting headache, change of the character, irritable disposition. Then follows, after a mental impression, *duppressio mensium*, or the proper attack. Also here was, in three cases, fever ; but catarrhs, etc., were mentioned as cause. During fever the mania has the character of the fever-delirium. Mairét distinguishes, further on, a "manie choréique simple" and a "manie choréique hallucinatoire." The simple form does not need to especially distinguish itself, in the way of appearance, from the common mania, but mostly there is noticeable a striking irritability, inclination to violence, emphatic manner of speech, and poetic scribbling as well as choreatic movements in different regions of the body. The excitement sometimes increases to repeated attacks of rage, during which the choreatic movements become more intensive, and springing, running, dancing impulsive acts, making of grimaces, and excessive masturbation form the prominent features.

With the hallucinatory form the deception of senses predominates and gives a corresponding feature to the delirium. They consist mostly in anxious imaginations and ideas of persecution ; the excitement may be of a milder

degree, as in the simple choreatic mania. The attacks of rage are also governed by the perceptions of the senses ; but the latter may also disappear, and the attacks take then the same course as does the simple form.

The choreatic mania may run on for months and even years. The prognosis is essentially more unfavorable as, in the case of lypemanic stupor, already because heredity plays a greater part in it. P.

Asylum Notes.

MATTHEW D. FIELD, M.D.

HOSPITALS FOR THE INSANE.

H. Hayes Newington, M.R.C.P.Ed., in his presidential address, delivered at the annual meeting of the Medico-Psychological Association of Great Britain and Ireland, takes for his subject "Hospital Treatment for Recent and Curable Cases of Insanity." After a review of public opinion and of recent articles that have reflected upon alienists, with reference to the cures now effected and the increasing numbers of the insane, he returns to the main subject of his address :

"It will be necessary first to point out that two distinct classes of hospitals are aimed at. The first is what we may call a county hospital, in which the cure of the patients admitted shall be the paramount object, the extent of scientific study depending on the aptitude of the staff. The second is the educational hospital, in which, while of course the good of the patient will not be lost sight of, the advance of science shall be the guiding principle. As the respective objects vary to a certain degree, so will the details of construction and service vary, and for this reason it is essential that the considerations of the two institutions shall be kept separate.

“Taking the county hospitals, the location thereof will be the first point to be settled. There can be no doubt that for the convenience of administration it must be within very easy distance of the main building. But it should not be immediately adjacent, or there will be danger that the central idea of separation from the ‘chronic’ will be prejudiced. It will be a great gain also if the main building were out of sight, so that new patients should not be reminded of the doom that may await them.”

The hospital accommodation should be equal to one-fifteenth part of the asylum population. This is arrived at by assuming that the average ratio of admissions into county and borough asylums to the average population thereof is about one to three and one-half. He would consider that sixty per cent. of admissions as *possibly* curable, and therefore suitable for admission into the hospital. “We may assume that a year’s residence should be the limit for doubtful cases, and that three months would be sufficient for the recovery of the most simple cases. Then the number of residents would be reduced by death, which, as we know, chiefly occurs soon after admission. It is extremely difficult to arrive at anything like certainty, but I should think that four months might be taken as about the average time which would elapse in a series of cases before either discharge or recovery, death, or transfer to the main building, on account of incurability, took place. This would give an average clearing of each bed in the hospital three times in the course of a year.”

“As to the arrangements of the hospital itself, only a few general suggestions can be made. Wards should find no place here. No room should be allowed to contain more than six patients at the outside, and where provision is made for association it should chiefly be for quiet melancholiacs, and, conversely, separation should be chiefly practised in cases of mania. No fact has been more impressed on my mind than that separation from other patients, and, indeed, from other people, tends to shorten and decrease excitement, and I can confidently say that not only the extent, but the nature even of an attack of acute

mania is beneficially influenced by comparative solitude. Therefore the proportion of single rooms should be considerable."

A small infirmary will be necessary. The furnishing and the decoration of the hospital should be more like that of an ordinary house than is the case in an asylum.

There should be provision for exercise, labor, and occupation of the patients. A separate garden, laundry and a small workshop or two solely for the inmates of the hospital are suggested, with simple games and other means of recreation.

The special attributes of a general hospital would be naturally provided for, such as laboratory, post-mortem room, and probably sooner or later a complete system of medical baths, including the Turkish bath, would be furnished.

The doctor who is head of the asylum should be also head of the hospital. "If possible it would be a great thing that the superintendent should be encouraged to avail himself of the aid of an outside physician."

The senior assistant medical officer should reside in the hospital.

The attendants should be chosen partly from without and partly from within the asylum. General hospital training being very essential, the wages of attendants should be relatively higher.

The second class of hospitals are then considered, and are styled educational hospitals. What was said as to the general structure and the subordinate staff applies here also.

"The matter of location will be determined by accessibility for teachers and students, as propinquity to a school of medicine will be the chief *raison d'être*. Probably London would be the first to erect such a hospital, but we should hope that if the experiment succeeded, which it would be bound to do, others would follow in Edinburgh, Glasgow, Dublin, and other educational centres.

"We have here no such guide to size as we have in the county hospital, since convenience of treatment must be the

measure. One hundred and fifty inmates should be, I think, the outside limit.

“The selection of patients would be matter of grave consideration. Selection it would have to be, for naturally a hospital built large enough to receive all the curable cases of a district which supports a medical school would be so large that the individual study of cases would be lost sight of. Nor should curability be an inflexible test, for obvious reasons. One rule, however, should be laid down, and that is that fancy or show cases should have the smallest possible representation; at all events for some time to come. I have adverted to the enormous difficulties in the study of mental disease, and it will be quite time enough to study recondite forms or varieties of disease when the mechanism of the simplest cases has been elucidated. Cases of so-called acute dementia, of simple mania and of simple melancholia, uncomplicated if possible with delusions, and certainly uncomplicated by organized or fixed delusion, should have the first and most liberal right of entry, and in these every endeavor should be made to connect general mental with special bodily abnormalities. Especial attention, for instance, should be given to the connection between the presence of morbid products in the blood, morbid heart conditions and blood pressures, with various forms of insanity, and so forth. Not that we do not know a good deal of these matters, but I suggest them as samples of the class of points on which the full weight of our new armament should be brought to bear. When such cases have been provided for, we should next choose some that are obviously connected with some well-marked bodily condition, such as the puerperal state, phthisis, syphilis, or gout. Then we may go back again to various forms of insanity marked by delusions. Next, we should open the doors to cases passing from acute to chronic, and we may well find room for a few chronic cases typical of the main varieties of insanity, for in studying the losing and the loss of intellect, we may reasonably expect to find traces of the conditions which cause the loss; and further, such cases are useful for comparison, and especially for the purposes of teaching. Finally, one or two carefully selected

specimens of each phase of general paralysis will complete a collection that seems to me to afford the most useful basis of study, both to the younger student and to the more advanced observer. It will be obvious that much careful adjustment will be required to insure such a selection as the above."

It is advocated that the staff be selected from alienists and general practitioners of medicine.

"But the adjustment of the various duties of both will be extremely delicate and difficult. In dealing with the insane there is one element whose influence is all-pervading and oppressive. It is the element of responsibility." This responsibility, he contends, must be in the hands of the alienist, and that the authority should rest with the resident portion of the staff, "precedent to the contrary notwithstanding."

"The circumstances of a hospital for the insane are quite different to those of a general hospital, and therefore the visiting staff, who in the latter would have extended authority, must be content in the former with the powers of teaching and treating, but not of administration."

The committee of management should be small. The senior resident medical officer should be a man far above an ordinary resident medical officer. "He should be of mature experience, should have had large responsible charge of the insane, should be of mature age and moral force sufficient to maintain under difficulties his position, without having constantly to fight to maintain his own; and, as a corollary, his stipend should be ample."

He should have a direct share in the clinical teaching and the power to regulate the independent clinical work of the students.

"Under him should be at least two assistant physicians, one of whom should be a very competent master of morbid anatomy. Resident pupils should also be provided for, to any extent which might be found convenient.

"Then as to the visiting staff. I should think that at least two alienists should have each a clinique assigned to them, and that each clinique should be a mixed one, that is

to say, the field of either should not be confined to one sex or to one form or class of mental disease. The visiting alienists should, of course, have full power to conduct the direct medical treatment of the cases submitted to them, but in matters involving a risk, such as the granting of leave of absence and final discharge, their authority must be subordinate to that of the resident physician."

The chief duties of the visiting alienists will be those of teaching, and this should be both systematic and clinical, and be conducted much on the lines of a general hospital.

The last and most difficult question will be the adjustment of the relations between the alienists and the general physicians. "Of these latter there should also be at least two, one skilled in all-round medicine and the other more especially in neurological conditions." Both should be on the staff of a general hospital, and both should have extended knowledge of physiology and its most recent advances. It will be their especial duty to endeavor to detect the least departure from normal bodily health, and they should work quite as much by the exclusion of the normal as by the following up of the abnormal. They should be required to conduct a thorough bodily examination of each case on admission, to renew these examinations from time to time, to order and supervise such methods of clinical inquiry as they may think fit, such as the use of the sphygmograph, quantitative and qualitative analyses, and so on. They would undoubtedly find a sufficiency of interesting material on which to found frequent clinical lectures. Then, too, they should cause the fullest and most exact records to be kept of their observations.

A well-conducted pathological laboratory and a large reference library of psychological medicine should be maintained. He also proposes an out-patient department, and believes that if these hospitals should be established that voluntary patients would be found to resort to them more readily than they would to present asylums.

GREAT BRITAIN.

REPORT OF THE COMMISSIONERS OF PRISONS.

The report of the Commissioners of Prisons for the year ending March 31st, 1889, shows the number of prisoners received during the year to have been 153,963 under sentence of the ordinary courts, 1222 soldiers and sailors sentenced by courts martial, and 8664 debtors, or imprisoned on civil process, making a total of 163,849. This shows an increase of nearly 7000 upon the numbers of the preceding year, and has occurred chiefly in prisoners under the sentence of the ordinary courts. The average daily number in prison was 15,255, of whom 12,673 were males and 2582 females.

There were 349 cases of insanity during the year, of which 210 were removed to asylums; 93 are stated to have been found insane upon reception, and 59 to have originated in prison. Of the other cases, it is said that "it is highly probable that although insanity was not immediately recognized, yet that it really existed on reception in a majority, if not the whole of them." Dr. Gover remarks: "The local prisons of England and Wales are at present used to some extent as hospitals for the treatment of mental and bodily disease, but as they are not intended for or adapted to that purpose, it is advisable that the practice in vogue should be discontinued as far as possible." We can hardly suppose that he means that convicted prisoners laboring under bodily disease should be exempted from undergoing their punishment of imprisonment on that account; and, as regards mental disease, the fact of 256 cases having occurred in which no such state was detected by the prison medical officer on admission justifies an opinion that the abnormal mental condition could scarcely have been ascertained before the prisoner was sentenced. Under such circumstances, it is difficult to understand how the prisoners could have been dealt with more satisfactorily in the interests of the public and for their own benefit—*The Lancet*.

The medical report of St. Luke's Hospital, Surrey, records a case of special interest, the wife of an officer in the army, who recovered after a residence of fourteen years. She left the hospital eight months since, and we have every reason to believe she continues well. There is some encouragement to persevere with treatment beyond the ordinary term of twelve months, in the fact that seven of the recoveries took place in the second year of residence.

It is also noted that the recent Whitechapel tragedies had a very determining character upon the nature of the delusions both in the men and women admitted from the East End of London. As a rule the delusions were of a temporary nature, and soon subsided—Dr. Shaw, in asylum report; Bansted, Middlesex.

Concerning the proposed new lunatic hospital in London, Dr. Howden remarks:—

Proposals have been made lately in the London County Council to establish hospitals for the treatment of cases of insanity, where the highest medical knowledge and skill will be brought to bear on the study and treatment of insanity. If this project be carried out, it will no doubt be an advantage to the Medical Schools by affording extended means for the study of mental diseases, but that it will be a benefit to the patients is, I think, more than doubtful, as I do not consider that any asylum can efficiently fulfil its curative functions unless it has plenty of land around it to afford scope for the occupation and exercise of the patients.

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